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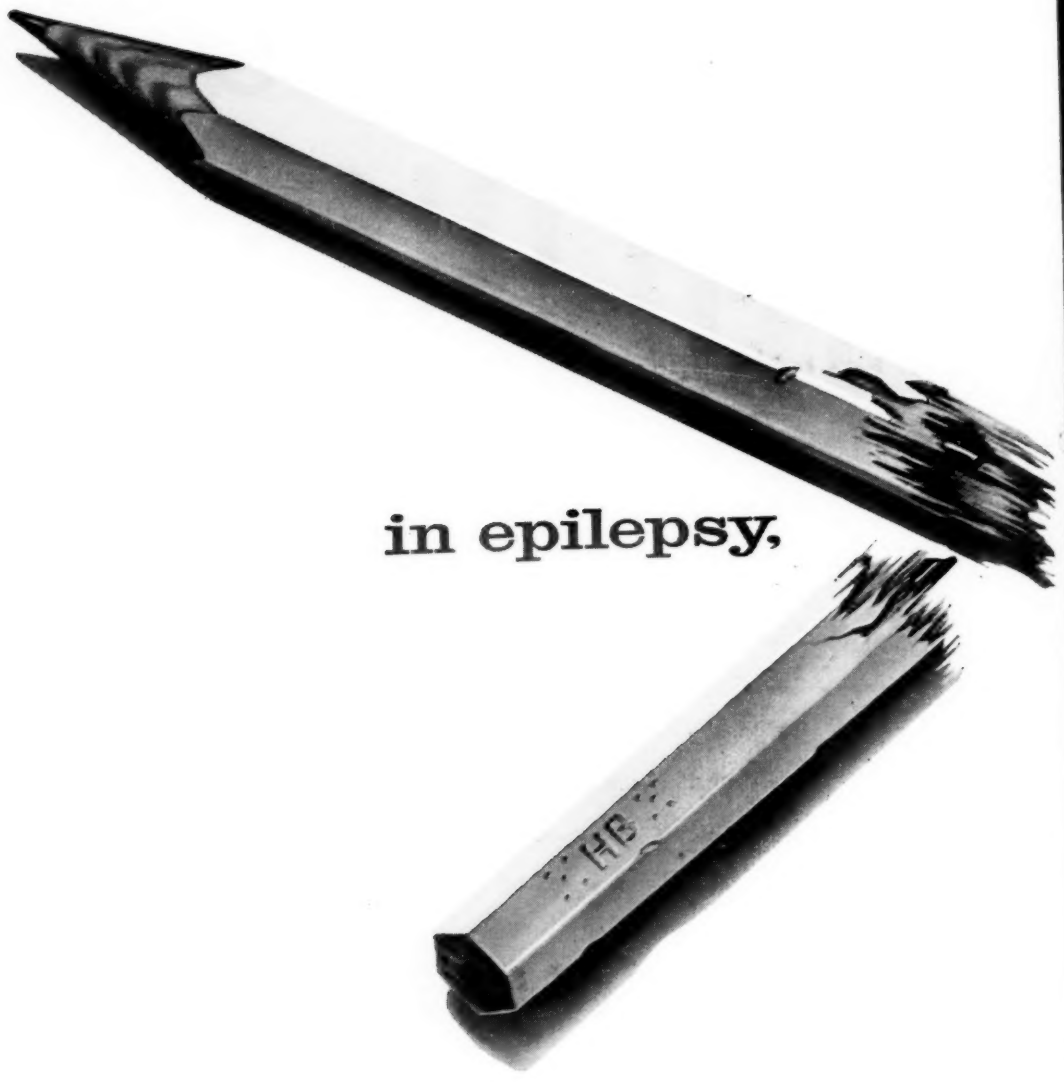
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# Minnesota Medicine

*Journal of the Minnesota State Medical Association, Southern Minnesota Medical Association, Northern Minnesota Medical Association, Minnesota Academy of Medicine and Minneapolis Surgical Society*

**Volume 37** **April, 1954** **Number 4**

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Minnesota Medicine

*Journal of the Minnesota State Medical Association, Southern Minnesota Medical Association, Northern Minnesota Medical Association, Minnesota Academy of Medicine and Minneapolis Surgical Society*

**Volume 37** **April, 1954** **Number 4**

# CANCER DETECTION

## A Report from the Cancer Detection Center, University of Minnesota

**CLAUDE R. HITCHCOCK, M.D., and J. BRADLEY AUST, M.D.**
**Minneapolis, Minnesota**

THE GOAL of the Minnesota Cancer Detection Center has been to develop a workable and accurate method of cancer examination including all internal organs as well as readily accessible external organs. Until a simple serodiagnostic or biologic test is available, such an effort is the only available means of detecting malignant disease in its early stages.

"Cancer Detection" implies the process of finding malignancies before symptoms or signs herald their presence, in addition to the diagnosis of precancerous conditions. Through the application of a well conceived routine plan of examination in doctors' offices, we believe several types of serious malignancy can be identified in advance of their usual signs and symptoms. More important than this, the 24 per cent of our population over the age of forty-five years who harbor a precancerous condition can be alerted for close observation or therapy.

During the five years from March 1, 1948, to February 28, 1953, cancer detection examinations have been performed on 5,352 men and women. Ninety-one cancers have been discovered at the Detection Center, and proved with biopsy or surgery. Treatment has been under the direction of the patient's personal physicians. Furthermore, a total of 1300 lesions considered to be precancerous have been detected and reported; an incidence of twenty-four per one hundred people examined over forty-five years of age. Table I demonstrates the ratio of new patient examinations and the incidence of malignancies and precancerous lesions found.

TABLE I.

| | Male | Female |
|---|---|---|
| New Patients | 2665 | 2687 |
| Recheck Exams | 3005 | 2986 |
| Total Examinations | | 11,343 |
| Total Patient's Examined | | 5,352 |
| Cancer Incidence | | 17 per 1000 patients examined (1.7%)<br>7.9 per 1000 examinations performed |
| Precancerous Lesions | | 1300 (24%) |

In order to facilitate the study and assure maximum cancer case finding a lower age limit of forty-five years was established. Recently the lower age limit for males has been raised to fifty years. The high incidence of carcinoma of the cervix in women in the fifth decade indicated the advisability of maintaining the lower age limit for females at forty-five years.

It is obvious that all people have *some* symptoms during any given interval of time. We have not been able to fulfill the original concept of obtaining a group of completely asymptomatic examinees for cancer detection observation. Therefore, our examinees with malignant disease have been separated into symptomatic and asymptomatic cases in reference to the particular organ or organ system in which the malignancy was found. In the group of ninety-one patients found to have cancer, forty-six patients had no symptoms referable to the organ or organ system in which the malignancy was detected and forty-five

Sponsored by the Council of the State Medical Society and Supported by a grant from the Minnesota Division of the American Cancer Society.

Dr. Hitchcock is Director, Cancer Detection Center, University of Minnesota, and Dr. Aust is Clinical Cancer Trainee, National Cancer Institute, National Institute of Health.

TABLE II. VALUE OF REPEATED RECHECK EXAMINATIONS IN DETECTING EARLY CANCERS

| | Asymptomatic | Symptomatic |
|---|---|---|
| New | 22 | 41 |
| Recheck | *24 | 4 |

*Note that over half of the lesions that were asymptomatic were found at recheck examination.

patients had symptoms referable to the organ or organ system involved. It will be shown later that survival figures are significantly better in the asymptomatic group. Table II demonstrates the value of the recheck examination in detecting asymptomatic cancers.

Table III portrays the cancers found in the first five years of operation, according to site and relationship to symptoms.

TABLE III. CANCERS FOUND IN FIVE-YEAR STUDY AT CANCER DETECTION CENTER

| Site | Asymptomatic | Symptomatic |
|---|---|---|
| Stomach | 6 | 5 |
| Colon and rectum | 13 | 5 |
| Small bowel | 1 | 0 |
| Lung | 0 | 3 |
| Breast | 6 | 1 |
| Ovaries | 2 | 3 |
| Cervix | 5 | 2 |
| Uterus | 0 | 2 |
| Prostate | 3 | 4 |
| Skin | 7 | 15 |
| Thyroid | 1 | 0 |
| Lymphosarcoma | 1 | 0 |
| Leukemia | 1 | 5 |
| Total | 46 | 45 |

### Method of Examination

Principles of completeness and economy have been important considerations in developing the pattern of the examination. Laboratory procedures have been reduced to bare essentials, requiring only a minimum amount of technical assistance.

A detailed history form is completed by the patient prior to his visit to the Center. The patient brings a urine and stool specimen (collected after three days on meat free diet) the morning of the examination. A routine urinalysis is performed (urine sugar, albumin, microscopic for cells if urine is positive for albumin). A benzidine test on the urine has been tried, but is not as accurate as microscopic cell study to detect occult urinary bleeding. The stool is examined for occult blood with the Guaiac and Benzidine test. Routine examination of the blood includes hemoglobin determination, white blood cell count, differential count and cell morphology.

Every patient has a Levine tube passed for gastric analysis using one dose of histamine (0.5 mgm.) as stimulant. The standard sodium hydroxide and Toepfer's reagent determination for free HCl has proved most satisfactory. The Quininium ion urinary excretion test has been tried and proved too time consuming in the laboratory for large numbers of determinations daily.

Chest x-rays are taken on each patient. Suspicious lesions or single nodules are further investigated with planigrams of the involved area. Sputum is studied for malignant cells by the Papanicolaou method. If no diagnosis is forthcoming, bronchograms, bronchial washings, and bronchial biopsy (if possible) are recommended.

All patients come to the Center prepared with enemas for a proctosigmoidoscopic examination. The scope is passed to the 25 cm. level, unless peculiar conformity of the bowel (acute angulation) or stricture prevents passage of the instrument. The following conditions are checked for: (1) small adenomas (2-3 millimeters); (2) polyps (sessile or pedunculated); (3) character of the mucosa (bleeds easily, inflamed, small ulcerations); (4) diverticula, diverticulitis; (5) hemorrhoids, fissures, fistulas; (6) frank ulcerating carcinomas or intramural tumors. Excision biopsy and fulguration is recommended for all small adenomas and polyps. During the digital examination of the rectum, the prostate is palpated for hypertrophy or nodules.

All women receive a pelvic examination including bimanual palpation of adnexal areas, and direct visual inspection of the vaginal vault and cervix. Any adnexal mass is considered suspicious in this age group, and pelvic laparotomy advised. Suspicious findings in the cervix includes: (1) rough area on lip of cervix; (2) polyps; (3) blood at the os, not menstruating); (4) atrophic cervical mucosa; (5) erosion of the cervix; (6) cervicitis.

Papanicolaou smears are made of vaginal secretions and scrapings of the cervix. Suspicious areas are biopsied. Positive Papanicolaou smears must be confirmed later by positive biopsy. Suspicious (Class III) smears, in an otherwise normal vagina, indicate need for repeated cytologic examination every six weeks until smears are

negative or biopsy proves the presence of a carcinoma.

After the laboratory work has been completed and the proctoscopic and pelvic examinations performed, a clinician examines the patient for the remaining problems:

1. Nose and throat with visual inspection of nasal cavity, oral cavity, and auditory canal. Inspection of the nasal pharynx and larynx with laryngeal mirror. Palpation of the neck and submandibular area for palpable lymph nodes. The thyroid gland is palpated for adenomas or masses.
2. Skin (senile keratoses, and pigmented nevi in areas of irritation are classified as suspicious). Any recently darkening or enlarging nevus is considered suspect for malignant melanoma.
3. Adenopathy is searched for in supraclavicular, axillary, and inguinal areas. The chest x-ray will detect mediastinal adenopathy.
4. Breasts are examined by inspection and palpation in both the sitting and supine position.
5. Abdominal palpation is important for detection of renal masses. Liver, spleen, gall bladder, enlarged lymph nodes, or any other masses are carefully checked.
6. External genitalia of the males are examined to include palpation of testes.
7. Peripheral muscle tissue and skeletal system are examined for deformities or lesions.
8. Brief neurological examination for possible manifestations of cerebral neoplasms.

## Criteria for Special X-Ray Studies

While summarizing the results of examination, the clinician determines whether further x-ray studies are needed according to the following plan.

*Gastric Intestinal Series X-Rays are Ordered for:*

1. Patients with histamine achlorhydria and hypochlorhydria (less than 30° free acid).
2. Patients with occult blood in the stool.
3. Patients with strong family history of gastric carcinoma.
4. Patients with unexplained hemoglobin level below 11 grams.
5. Patients with vague symptoms of gastric pathology which in any sense could be interpreted as being due to gastric carcinoma by the examining physician.
6. Any evidence of undue weight loss.

The air contrast technique is helpful in cases of suspected gastric polyps or small mucosal defects.

*Barium Enema Colon X-Rays are Indicated for:*

1. Patients with abnormal findings on proctoscopic examination.
2. Patients with occult blood in the stool.
3. Patients with unexplained hemoglobin level below 11 grams.
4. Patients with a family history of large bowel malignancy.
5. Patients who have vague symptoms that could possibly be due to malignancy of the large bowel as determined by the examining physician.
6. Any evidence of undue weight loss (10 pounds or more).

TABLE IV.
COMPARISON OF GASTRIC CANCER PATIENTS

| | Asymptomatic | Symptomatic |
|---|---|---|
| Total Number | 6 | 5 |
| Achlorhydria | 6 | 5 |
| Occult Blood | 1 | 1 |
| X-Ray Diagnosis (1st Visit) | 1 | 2 |
| X-Ray Diagnosis (Subsequent Visits) | 5 | 3 |
| Scirrhous | 1 | 3 |
| Polypoid | 5 | 1 |
| Ulcerating | 0 | 1 |
| Positive Lymph Nodes | 1 | 4 |
| Alive | 3 (1½, 3, 5 yrs.) | 1 (1½ yr.) |
| Deceased | 3 (1¼, 1/12, 1 yr.) | 4 (1/12, ½, ½, 1 yr.) |

*Survivals are considered as of December 31, 1953.

In addition to routine barium enema x-rays, all patients found to have polyps on proctoscopic exam are given air contrast barium enema studies.

*Intravenous Pyelogram X-Rays are Ordered for Patients with:*

1. Blood in the urine.
2. Costovertebral pain or tenderness.
3. Palpable mass in region of kidney.

## Results of Five Years' Operation

*Esophagus and Stomach.*—Eleven gastric carcinomas have been found, seven in males and four in females. All patients had achlorhydria and would have been been picked up by x-ray performed on this basis alone exclusive of other indications for performance of a G.I. series. In only two cases (with symptoms) was the hemoglobin below 11.0 grams. Occult blood was present in the stool of only one asymptomatic patient and one patient with symptoms. Table III demonstrates that in six patients the gastric cancers gave no symptoms, while in five patients symptoms were present.

Polypoid type carcinomas were found in *five* of the *six asymptomatic* patients and scirrhous carcinoma in one patient. In the *five* gastric cancer patients with symptoms, *four* had scirrhous carcinoma, and only one had a polypoid lesion. Table IV demonstrates the results of early diagnosis (asymptomatic patients) compared with late diagnosis (symptomatic patients) in the gastric cancer group. Three of the patients are living and well up to five years in the asymptomatic group. There is only one survivor in the group of patients with symptoms at time of diagnosis.

Early polypoid lesions have a good chance for cure with subtotal gastrectomy. Since virtually 100

TABLE V. RECTAL AND COLON CANCERS

| | Asymptomatic | Symptomatic |
|---|---|---|
| Total Number | 13 | 5 |
| Diagnosed first visit | 11 | 5 |
| Occult blood | 2 | 3 |
| Hemoglobin below 12.5 | 0 | 1 |
| Proctology positive | 10 | 4 |
| Barium enema | 3+ (also 7 of 10 procto pos.) | 3+ (also 2 of 4 procto pos.) |
| Ulcerating cancer | 2 | 4 |
| Polypoid cancer | 11 | 1 |
| Lymph nodes positive | 2 | 3 |
| Alive | 11 (2½, 2½, 4, 3½, 4¾, 2¾, 1¾, 3¼, 5¼, 4¾, 4¾ yrs.) | 1 (2¾ yrs.) |
| Deceased | 2 (1¾, 2 years after CVA) | 4 (1/12, 7/12, 1¾, 2½) |

*Survivals are considered as of December 31, 1953.

per cent of polyp-bearing stomachs are achlorhydric, the gastric analysis seems to be a satisfactory method for indicating those patients with a likelihood for polyps. Polyps greater than 2 cm. in diameter have a 50 per cent chance of being malignant when found. Of those 1 cm. or less in diameter when detected about 11 per cent are malignant.

For early asymptomatic gastric cancers the most significant criterion indicating need for gastrointestinal series x-rays is achlorhydria. We do not know of a stomach cancer that has been found during this five years among the three-fourths of our patients who have normal gastric acidity.

One examinee presented during the first six months of operation of the Center with total obstruction of the esophagus due to known esophageal carcinoma. He was too ill to undergo the examination and is not counted in the series of cancers picked up in the Center. This was the only esophageal cancer seen during the first five years of function of this Center.

*Colon and Rectum.*—Eighteen cases of cancer of the rectum and colon have been detected: Ten in males and eight in females. Fourteen of the lesions (77 per cent) were within reach of the sigmoidoscope. Thirteen of these fourteen were in the 9 cm. to 16 cm. level. Three lesions were in the ascending colon or cecum and one was in the descending colon.

Of the eighteen patients, thirteen were free of symptoms for G.I. tract disease and five had symptoms referable to the G.I. tract. Only two of the thirteen asymptomatic patients had occult blood in the stool; three of the patients with symptoms had occult blood. Here again, for early lesions still not causing symptoms, the stool occult blood determination is not a satisfactory single screening device for indicating necessity of x-ray examination.

Routine examination of the rectum and sigmoid through the sigmoidoscope has proved to be the most useful screening device for large bowel lesions. Table V compares the colon and rectal cancers with and without symptoms. Of the group of thirteen symptom-free patients, eleven are living and well up to five years (seven over three years). Only one of the five patients who had symptoms referable to the colon at the time of diagnosis is living and well (two and three-fourths years).

There is no etiologic relationship between diverticulitis and carcinoma of the colon. However, inflammatory strictures due to diverticulitis often cannot be differentiated from constricting adenocarcinoma by x-ray studies. Exploratory laparotomy and resection of the involved area should be performed for potentially obstructing inflammatory lesions, thereby circumventing the risk of unsuccessful differentiation of the benign from the malignant process.

*Female Pelvic Organs.*—Seven cancers of the cervix have been found and proved. Five women were without symptoms and two had symptoms of pelvic disease when examined. Two women in the asymptomatic group had negative Papanicolaou smears, but suspicious areas noted during inspection of the cervix indicated the need for biopsy. In only one case (asymptomatic) was the cervix entirely normal in appearance and the positive Papanicolaou smear was the specific indication for biopsy which proved the presence of the carcinoma.

A total of 7,940 Papanicolaou studies have been performed. This includes both vaginal and cervical smears. Ninety-eight Class III (suspicious) reports were made, and ten Class IV (malignant cell) diagnoses were made. Seven of the above 108 patients had squamous cell cancer proved and treated. It is possible that an indeterminate number of the patients in whom a suspicious Papanicolaou has been reported, will eventually prove to have cervical carcinoma.

However, it must be emphasized, that visual inspection of the cervix with biopsy of any sus-

picious areas provides an excellent chance of detecting lesions early; particularly when such examinations are repeated at yearly or six-month intervals.

*Endometrial Carcinoma.*—Two endometrial carcinomas have been found, and both had symptoms and signs of disease (abnormal spotting). Papanicolaou smears were negative. Diagnosis was established by dilatation and currettage. Both patients are living and well after surgery. No good screening technique is yet available for *early* endometrial carcinoma. Bleeding and discharge from the cervical os, and uterine enlargement remain well-proved danger signs.

*Ovarian Carcinoma.*—Of forty-seven women who reported back to their physicians with a diagnosis of suspicious adnexal masses, five proved to have carcinoma of the ovary. Four of these patients were asymptomatic and one had symptoms. All were detected by means of bimanual palpation. In every case the surgical specimen revealed early cystadenocarcinoma of the ovary without spread to other tissues. All patients are living and well. Pelvic examination is the only method for detection of these early ovarian tumors. Laparotomy is necessary to establish the nature of a palpable mass.

*Prostate.*—Thirty-two patients were reported to have prostatic nodules suspicious for carcinoma. Twenty-one had resection or biopsy of the lesion, and seven carcinomas were proved histologically. We have accepted only histologic proof of malignancy, as against clinical impression. Nine patients have been treated for carcinoma of the prostate on clinical evidence only. It is curious that of all human malignancies, the diagnosis of prostatic cancer is commonly accepted on clinical evidence alone, whereas histologic or cytologic proof is demanded for other forms of cancer.

Three of the seven proved cases were asymptomatic, and four had symptoms of prostatic obstruction. Two of the symptom-free patients had bony metastases, as well as two of the patients with symptoms. One out of the four symptomatic patients is dead, and two of the three asymptomatic patients are dead. No satisfactory screening test is available for prostatic cancer. Unfortunately, widespread bony metastases are found in many patients at the time symptoms of prostatic obstruction appear. It is only conjectural to assume that repeated examination will significantly alter the outcome of prostatic cancer.

*Skin.*—Twenty-two patients have had skin cancer diagnosed at the Center. Fifteen of the lesions were basal cell carcinomas, and six were squamous cell carcinomas. One pigmented nevus proved to be a malignant melanoma. All of these lesions were small and localized; treatment has been successful, and all of the patients are well at this time. Fifteen of the twenty-two patients were aware of the presence of the lesion, but considered it benign and of no consequence.

Senile keratosis of the skin is considered a pre-malignant lesion and 183 patients have been found with keratoses. A total of 240 pigmented nevi, exposed to chronic irritation, have been reported, but only the one malignant melanoma has been demonstrated. No problem attends the diagnosis of skin cancer when clinicians are quick to submit biopsies of all suspicious lesions for histologic evaluation.

*Lung.*—Only one primary carcinoma of the lung was found in the first five years of function of the Detection Center. This lesion was far advanced and the patient had obvious symptoms of pulmonary malignancy. Two metastatic carcinomas were picked up, one a metastatic melanoma (not our melanoma patient) and the other a metastatic hypernephroma. No case of asymptomatic primary carcinoma of the lung has been detected, but one examinee was misdiagnosed, and will be reported in a following section. This is an unusual circumstance, perhaps owing to the exceptionally good detection of lung lesions in the mobile unit chest survey which has been extensively used in our State. Willingness on the part of our examinees to participate in a cancer detection program indicates a likelihood that they availed themselves of the opportunity for a chest x-ray examination.

There are no pathognomonic symptoms of *early* lung cancer. When symptoms are present during the earliest *phases* of lung cancer, they are the same as those produced by common respiratory diseases. These symptoms often respond to non-specific treatment, and serious delays in accurate diagnosis and treatment are fostered. The highly lethal nature of lung cancer makes it imperative that clinicians exercise a

high index of suspicion towards silent lung nodules (coin lesions) and other suspicious pulmonary changes if a significant increase in five-year survivals is to be effected.

*Breast.*—Ninety-two women had breast lesions considered suspicious for the presence of cancer. Seven (9 per cent) carcinomas have been proved at surgery, and seventy-seven lesions have been established as benign. Eight lesions are not yet fully diagnosed. All of these tumors were unknown to the patients at the time of examination.

Six patients are living and well, and one is dead of cerebral metastases fifteen months after surgery. At the present time periodic physical examination of the breasts is the only proved method of detecting this cancer.

*Urinary Tract.*—The kidney has been a difficult organ to survey with detection techniques. Routine urinalysis for detection of urinary tract bleeding has not been adequate as an indication of the need for advanced roentgen studies. Most kidney lesions are large enough to be palpated during physical examination before they have invaded the renal pelvis and caused urinary tract bleeding. The Detection Center missed the diagnosis in two patients who subsequently developed adenocarcinoma of the kidney. In the absence of an adequate screening technique, and with widespread use of roentgen pyelogram unfeasible, we probably will not improve in diagnosing these patients in an asymptomatic stage of the disease. No testicular or penile cancers have been found.

*Lymphatic System.*—Of six patients found to have lymphatic leukemia, four had palpable lymph nodes at the time of examination. Only one was asymptomatic. Five had symptoms of a mass, pain or weakness. There were forty-six patients with a lymphocyte count above 45 per cent, and thirty-nine patients of this group have not developed a lymphoma to the present time. Four of the six leukemia patients are alive up to five years after treatment.

*Thyroid.*—One papillary adenocarcinoma of the thyroid was proved at surgery, and one lymphosarcoma of the thyroid was found. These glands were diagnosed as the sites of suspicious nodules. A total of eighty-six thyroid adenomas have been reported. Thirty-five of these have been surgically excised. We believe about seven per cent of palpable, single, thyroid nodules will prove to be malignant (University of Minnesota study).

TABLE VI. PRE-CANCEROUS LESIONS

| Total Number | 1300 |
|---|---|
| Adenomas and polyps of rectum and colon | 624 |
| Gastric polyps | 6 |
| Kraurosis vulvae | 16 |
| Leukoplakia | 144 |
| Pigmented nevi (areas of irritation) | 240 |
| Polyps of vocal cords | 1 |
| Senile keratosis | 183 |
| Thyroid adenomas | 86 |

*Pre-Cancerous Lesions.*—Table VI presents eight lesions considered to be pre-cancerous and the number of patients detected with these conditions. It is impossible to calculate the potential salvage in this large segment of the over-all group (24 per cent) through *prevention* of malignancy by application of appropriate therapy during the pre-malignant stage of such lesions.

*Known Failures of the Detection Center Program During Five Years Operation.*—It is to be expected that a few cancers might arise in such a manner that detection center procedure would not screen out the lesion, or that failure of an examinee to co-operate would result in late diagnosis. Furthermore, an occasional misinterpretation of roentgenograms might result in undue delay in establishing the histologic nature of a questionable lesion. Table VII presents nine examinees who developed cancer during the interim period between examinations, and were not picked up at the last negative examination.

Two patients with carcinoma of the kidney were asymptomatic at examination two months and ten months prior to diagnosis. Urinalyses were negative, and abdominal palpation was negative; criteria indicating the need for intravenous pyelograms were lacking. Since intravenous urography is not feasible as a routine procedure, and since most renal neoplasms in adults attain considerable size before invasion of the renal pelvis causes symptoms or signs of disease, a better method of screening for renal neoplasms is urgently needed. These patients demonstrate an inadequacy of the present examination procedure for renal lesions.

The gastric cancer appeared in a patient who previously had a subtotal gastrectomy for duo-

TABLE VII. KNOWN DETECTION CENTER FAILURES DURING FIVE YEARS OPERATION

| | Time from CDC Exam. to Positive Diagnosis | Conditions |
|---|---|---|
| Kidney | 2 months (surgery) | Urine negative. Prostate questionable. Recheck by LMD disclosed renal lesion. Failure of examining method. |
| Kidney | 10 months (surgery) | Examination and urinalysis at CDC negative. Failure of examining method. |
| Stomach (carcinoma) | 25 months (surgery) | Subtotal resection 1923 for duodenal ulcer. X-rays and exam. at CDC negative. Patient refused follow-up exams by CDC & P.P. |
| Sigmoid (carcinoma) | 4½ months (surgery) | Exam. at CDC diverticulitis. Exam. by LMD same. Then developed bleeding and procto biopsy positive. Failure of examining method. |
| Left Lung (carcinoma) | 18 months (surgery) | Jan. 1949—apical lesion seen here. Diagnosed fibrosis. Jan. 1950—diagnosed same. Summer 1950—carcinoma. |
| Tonsil (undifferentiated carcinoma) | 8 months (biopsy) | Negative exam. at CDC 8 months prior to symptoms. |
| Neck (lymphosarcoma) | 8 months (died) | No indications 6 months prior to death. |
| Prostate (carcinoma) | 14 months (died) | Previous trans-urethral resection for prostatism, 1946. Died of heart disease; cancer prostate incidental finding. |
| Unknown (abdominal carcinoma) | 14 months (died) | 1949—Papanicolaou Class III—biopsy of cervix negative. Exam. 1950 negative. Failure recheck appointment. |

denal ulcer. Gastrointestinal series x-rays were negative for tumor at the time of the CDC examination. The examinee failed over a twenty-five-month period to return to either the Cancer Detection Center or his own physician for check-up. It is reasonable to assume that the gastric malignancy developed during that period of time, for the silent interval of most gastric cancers is in the nature of twenty to twenty-two months. Patient failure appears to be the responsible factor in this instance.

The carcinoma of the sigmoid represents a failure to correctly interpret the colon roentgenograms. A diagnosis of inflammatory stricture secondary to diverticulitis was made at the Cancer Detection Center, and confirmed with further x-ray studies by the family physician. An episode of bleeding shortly after the second x-ray examination led to biopsy of the area through a sigmoidoscope and the maligant character of the constriction was established. The roentgenologist must exercise caution in attempting to differentiate between lesions of the colon causing stricture. Laparotomy with excision of the diseased part is the safest course to follow in any case of large bowel stricture.

The lesion of the left lung was noted on two chest films taken at the Cancer Detection Center one year apart. Six months following the second diagnosis of pulmonary fibrosis the patient developed symptoms, and chest x-ray showed progression of the lesion. Thoracotomy then established the malignant character of the tumor. Failure to strongly advise thoracotomy rests with the Detection Center. Pulmonary lesions must be established histologically at thoracotomy if sputum studies and bronchoscopic biopsy fail to establish the diagnosis. Undue delay is particularly tragic with this lethal carcinoma.

One carcinoma of the tonsil and one lymphosarcoma of the neck appeared eight months after a negative examination at the Detection Center. There was no evidence of tumor in either patient at the time of examination at the Detection Center. Another patient was found to have carcinoma of the prostate as an incidental autopsy finding following death due to cardiac disease. The last patient died of carcinomatosis fourteen months after a negative examination. The source of the primary tumor was obscure at autopsy.

A recent survey of examinees who failed to return for recheck examinations has not brought other cases of "missed cancers" to our attention. This latter group represents 9 per cent of the hundred cancers known to be present in the entire five-year period.

Of the ninety-one cancers correctly diagnosed in the Cancer Detection Center, thirty-two were established by roentgenograms of one kind or another. Only two of the "missed" cases were examined with x-ray methods and these constitute 6 per cent failure for roentgen ray diagnosis during the five years of operation of the center. This compares favorably with diagnostic experience in the x-ray department of the University of Minnesota Hospitals. All roentgen diagnostic methods have specific limitations, and clinicians responsible for patients must appreciate the necessity of bringing other more specific methods (microscopic examination of tissue) to bear on questionable cases.

Sixty-nine of the cancers correctly diagnosed were found either during physical examination or with a laboratory test. If we consider six of the "missed" lesions as failures of physical diagnosis, this represents 8 per cent inaccuracy as far as missing malignancies is concerned. It must be remembered, however, that many lesions were

reported as suspicious (prostate, ovary, breast, thyroid) and proved to be benign. Again, the shortcomings of gross palpation or inspection are obvious, and the necessity for histologic and cytologic verification is amplified.

## Summary

1. During five years' operation of the Cancer Detection Center at the University of Minnesota ninety-one malignancies were found in 5,352 examinees. A total of 11,343 examinations were performed. This represents seventeen cancers per 1,000 patients examined (1.7 per cent), or 7.9 cancers per 1,000 examinations performed.

2. Forty-six examinees with cancer were symptom free for the organ or organ system in which the lesion was found. Forty-five had symptoms indicating a likelihood of cancer in the organ or organ system involved. In the asymptomatic group twenty-four cancers were found at recheck examination, indicating the value of repeated examinations to detect malignancies in their earliest stages.

3. Only in cancer detection centers, or cancer prevention clinics are large numbers of persons surveyed for cancer while they are without symptoms of disease. It seemed of value to emphasize the difference in survival between symptomatic and asymptomatic examinees found to have cancer in this study.

4. Three of six asymptomatic gastric cancer patients are alive and well up to four years after surgery. There is one survival among five gastric cancer patients who were symptomatic at the time of diagnosis. Of thirteen patients with colon cancer who were free of symptoms at time of diagnosis, eleven are living and well (seven over three years). Only one of the five colon cancer patients with symptoms at time of diagnosis is living (two years). These results appear to substantiate the increased value of early diagnosis in terms of survival.

5. Twenty-four per cent of our patients had pre-cancerous lesions. The value of treatment of these lesions in the prevention of cancer is incalculable.

6. Of all cancers developing in our examinees during this five years, 9 per cent are considered failures of the Detection Center method. Roentgen ray techniques failed in 6 per cent of the thirty-four patients in whom the diagnosis was established by this modality. Misinterpretation of x-ray changes was the responsible factor. Of the cases detected by means of physical examination and laboratory studies 8 per cent must be considered as failures for this method.

7. We believe that several types of serious malignancy can be identified in advance of their usual signs and symptoms through the application of a well conceived, routine, cancer detection examination performed at yearly intervals.

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*University of Minnesota Hospitals*

# FARMER'S LUNG

## A Form of Bronchopulmonary Moniliasis

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IN RECENT MONTHS some instances of chronic pulmonary disease in farmers have been observed which share certain features in common. Each gave a history of uncomfortable exposure to the dust from moldy silage, each was ill for a period of months complaining of dyspnea or cough, and each improved on removal from his environment.

### Case Reports

*Case 1.*—The first man was a dairy farmer forty-four years old, who presented himself in April, 1953, with the complaint of abdominal soreness and constipation associated with loss of ambition and a 20-pound weight loss of four months' duration. Physical examination disclosed a few palpable posterior cervical lymph nodes, and some tenderness in the epigastrium and right lower quadrant of the abdomen. The blood hemoglobin was 12.9 Gm., and the white cell count was 8,400 per cu. mm. with 77 per cent neutrophils, 12 per cent lymphocytes, 5 per cent monocytes, and 6 per cent eosinophils. The Wasserman test gave negative findings. The histoplasmin and tuberculin skin test findings were also negative. X-ray examination of the gastrointestinal tract showed only tenderness and spacticity over the duodenal bulb and colon. The chest x-ray examination revealed blunting of the costo-phrenic angles, a small amount of fluid in the right major interlobar fissure, and a mottled infiltration of both mid-lung fields extending out to the periphery. Upon questioning, the patient stated that he had been experimenting with a method of storing silage dry in the open field. He was quite disgusted with the method because every time he went out to the field storage area to get a load of silage for his cattle, he was distressed with suffocating chest pain after inhaling the dust from this moldy silage.

He also stated that two other farmers in an adjoining county had been storing silage in the same manner, and had noted respiratory distress following inhalation of the dust. They had been told at the University of Wisconsin Hospitals that they were probably allergic to something in their silage. When seen four months later, eight months after the onset of his illness, he felt improved, but he had done no farm work in the interval. The chest radiograph showed resolution of the previously noted infiltration into streaks of small nodular densities radiating out from the hilum.

*Case 2.*—The second case was that of a dairy farmer forty-six years old, who began to note a productive cough and shortness of breath in the fall of 1953. By the end of the year, he was unable to do any work and at times required adrenalin by injection to relieve his nocturnal dyspnea. Treatment with several different antibiotics given one after the other for two to four weeks at a time apparently gave him little relief from his symptoms. No notes concerning the physical examination during the acute phase of the illness were available from the referring physician, but when the patient was first seen in May, 1953, he was rather pinched and anxious in appearance and had a slate gray cyanosis of his skin and mucous membranes. There were no expiratory wheezes audible, but on inspiration there were many sticky and crackling râles. Blood hemoglobin was 18.5 Gms. per cent and the white cell count was 8,550 per cu. mm. with 75 per cent neutrophils, 13 per cent lymphocytes, 9 per cent monocytes, 2 per cent basophils, and 1 per cent eosinophils. The sputum was viscid and contained non-hemolytic streptococci, coagulase negative staphylococci, and some eosinophils. No fungi were demonstrated by smear or culture. Tuberculin and histoplasm skin test findings were negative. X-ray examination of the chest at monthly intervals, commencing in January, 1953, had shown a finely mottled type of density distributed widely through both lung fields. By May, 1953, there was some clearing of the widespread infiltrations without coalescence of nodules or formation of fibrous residuals. This man stated that he had not finished his new silo in the fall of 1952, and had to rely on silage left standing in the cattle barn or trucked into the barn from neighboring farms. Inhalation of the dust from this silage caused so much dyspnea and cough that he was unable to carry on through the winter with the daily chore of pitching of silage out to the cattle. This work was finally delegated to his wife who seemed to handle the job without much trouble.

*Case 3.*—The third case is that of a farmer, sixty-four years old, who stated that he had noted considerable respiratory distress after handling moldy hay over several winters. He felt that he was suffering from repeated upper respiratory infections. In March, 1953, he noted the onset of a cough productive of two or three ounces of thick yellow sputum daily. Dyspnea on slight exertion was severe, and there were frequent wheezing respirations. On physical examination crackling râles were noted over both lungs, and there was cyanosis of the lips and nail beds. The remainder of the examination was without note of abnormality. Blood hemoglobin was 14.7 Gm. per cent and the white cell count was 6,450 per cu. mm. with 72 per cent neutrophils and 2 per cent eosinophils. Sputum smears and cultures failed to demonstrate pathogenic bacteria or tubercle bacilli, but Candida albicans was isolated from sputum culture on three occasions and a penicillium on one other occasion. X-ray examination of the chest showed finely mottled densities scattered through both lung fields. Over a period of

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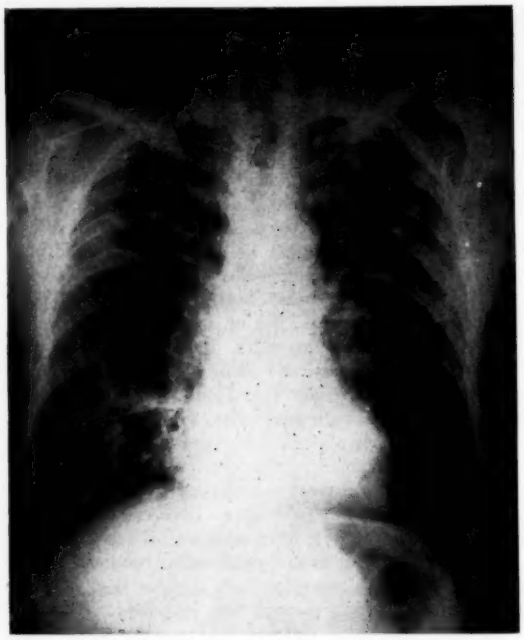
two months in the hospital, there was noticeable clearing of the diffuse infiltration, and the dyspnea was less severe than on admission.



Roentgenogram of chest in third case* after clearing of the acute pneumonia. There are widespread fibronodular infiltrations radiating from each hilum.

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## Discussion

These three cases of idiopathic pneumonitis, or pneumoconiosis of farmers, had some features obviously in common, and seemed to resemble those cases of "farmer's lung" recently summarized from the British and Scandinavian literature by Fuller.[3] In England, roentgenologists have been describing an occupational pneumonitis among farmers for over twenty years.[2] They have recognized in farmers exposed to the dust of "burnt" hay or moldy grain at threshing time a bronchitis which may go on, after repeated exposure, to a kind of bronchopneumonia, and, after many years of repeated exposure, to a fibro-cavitary disease roentgenologically indistinguishable from tuberculosis. A high spore count for molds of the Fungi imperfecti was obtained from the airborne dust in either the cattle barns or from the threshing floors where these people worked. In most instances, the Monilia, Candida albicans, was recovered from the sputum of the infected workers.

Whether or not these cases represent instances of primary pulmonary moniliasis is certainly open to question. There is little doubt but that primary pulmonary moniliasis can occur, but it is extremely difficult to make the clinical diagnosis of that disease.

Castellani,[1] in 1905, described "tea-tasters cough" and pneumonitis in persons employed in the dusty tea warehouses of Ceylon. Tea-tasters crushed the dried tea leaves in their hands before sniffing them for aroma while coolie laborers carried bags of dried leaves around within the dusty warehouses. Both groups developed a peculiar cough, and it was known that laborers who became ill while working in the warehouses would recover if employed in some outside work for a time. Candida albicans was found in the dried tea leaves, was recovered from the sputum of sick men, and was made to reproduce the disease in animals. This last was accomplished by forcing a guinea pig to inhale daily over an eleven-month period the tea leaf dust contaminated with Candida albicans.

A brilliant demonstration of the pathogenicity of certain strains of Monilia was made by Dr. Kano Ikeda[4] of Saint Paul and was reported in 1936. He collected five cases of bronchopulmonary moniliasis occurring both as part of systemic moniliasis in debilitated persons as well as primary pneumonia in young individuals exposed to a dusty atmosphere. One particularly virulent strain was isolated from a young man with pneumonia and empyema due to Candida albicans. When this organism was inoculated into rabbits by the intravenous route, they died within a few days of uremia. When the inoculation was intratracheal, a bronchopneumonia developed. When the inoculation was made into the lung by a direct stab through the chest wall, the injured lung offered little resistance to infection and a severely destructive pneumonia resulted. Ikeda also noted the presence of both sub-miliary tubercles and obstructive thickening of the walls of smaller arterioles in the inoculated rabbit lung as well as the human autopsy material he examined. This is particularly interesting in the light of recent work performed by Lennart Zettergren in Sweden.[5]

In 1950, Zettergren reported on an experiment to determine the rôle played by organic dust in

the causation of bronchopenumonia in persons working on threshing rigs. He found the dust from a threshing machine to be heavily infested with Candida albicans. The monilia organism was propagated on wort agar while the dust was sifted and sterilized. A chamber was then constructed in which a rabbit could be confined in an atmosphere heavily contaminated with sterile threshing dust. The first group of rabbits was exposed for an hour a day for fourteen days to the dust alone, and the lungs were then examined as the animals were sacrificed at intervals of a few weeks following exposure. A second group of rabbits was exposed to the sterile dust innoculated with the monilia culture, and the lungs were then examined in the same manner as with the first group. In both groups of animals, there was evidence early of irritative bronchitis due to the dust. Both groups showed submiliary granulomata in the neighbhood of the terminal or respiratory bronchioles. These granulomata persisted only a few weeks in the animals exposed to dust alone, but showed caseation and persisted much longer in the animals exposed to dust and the fungus. In addition, the second group of exposed animals showed thickening of blood vessel walls in the terminal arterioles, some being almost obstructed. No fungi were demonstrated in the granulomata, but numerous fat-filled macrophages were seen in the caseating granulomata.

The most interesting thing about Zettergren's descriptions of the pathologic changes in the rabbit lungs was that it was almost identical with Dr. Ikeda's descriptions of the changes in both human and rabbit lungs reported fifteen years ago, although there is no indication that Zettergren was familiar with Ikeda's report. It is apparent that the granulomata accounted for the miliary picture seen on the chest radiograph and it seems reasonable to assume that the arteriolar obstruction accounted for the pulmonary congestion and the cyanosis noted in the subacute phase of the reported cases. The hemoglobin value of 18.5 Gm. per cent in one of the reported cases and a similar elevation in an unreported case seen at the University Hospital since these cases were collected is conceivably a compensatory polycythemia in response to the chronic hypoxia caused by the arteriolar obstruction.

These cases are not presented as authenticated instances of pulmonary moniliasis. As previously stated, diagnosis is extremely difficult to make in the acute phase of the illness and is nearly impossible to make in the chronic state except by lung biopsy and tissue culture. However, these cases do fulfill the requirements for making a presumptive diagnosis of pulmonary moniliasis, each patient having been exposed to an irritating dust from a moldy source and each demonstrating the roentgenological and clinical picture that has been associated with proven cases of pulmonary moniliasis.

Ordinarily, in our temperate climate, we should not expect to find much monilial infection, but with the unusually wet field conditions of recent years, we should anticipate much greater exposure to fungi among our agricultural workers. Mycotic pneumonitis must be considered in any case of persistent pulmonary disease in farmers. Since the fungi grow so freely after antibiotic therapy in such a large percentage of people so treated, it becomes increasingly important to identify pathogens before instituting therapy for pneumonitis. In unusual cases, the bronchoscopic aspiration of secretions is essential to by-pass the usual contaminants in the upper respiratory tract when material is needed for diagnostic culture. It is to be hoped that when atypical pneumonitis in farmers is encountered in the future that interested persons may seize the opportunity to identify the causative organisms and trace them to their source. This procedure will reward the investigator because if fungi are found to be causing the pneumonitis, they will frequently prove susceptible to iodides whereas antibiotics will have little effect and may even enhance the growth of fungi.

Farmers certainly should be dissuaded from employing farm practices that result in the propagation and dissemination of moldy dust, for the pulmonary mycoses are extremely debilitating and may leave permanent residuals, if exposure is repeated.

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# UPPER GASTROINTESTINAL HEMORRHAGE

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A FORTY-FIVE-YEAR-OLD man was first seen in June, 1950, with a history of incomplete recovery from "flu." His complaints were of minor, indefinite aches and pains in the chest and epigastrium. His general appearance and health were good. He did have a past history, however, of excessive use of alcohol. The general physical examination and routine blood studies, as well as x-ray examinations of the chest, colon, esophagus, stomach, and duodenum, all gave negative findings.

Five weeks later, he became weak and faint and was admitted to his local hospital. The next day he vomited blood and had two large bloody bowel movements. During the following three weeks, he had repeated massive upper gastrointestinal hemorrhages requiring thirty-five transfusions. He was seen by many consultants from whom came a variety of opinions. His condition prevented operative treatment and he expired. A small ulcer high on the lesser curvature was disclosed at a postmortem examination. In its base there was a small ruptured aneurysm of a branch of the left gastric artery. A thrombotic mass with signs of organization was attached to the intima of the most superficial part of the blood vessel.

This case and repeated experiences with sudden severe gastrointestinal hemorrhage stimulated a study of such cases admitted to St. Mary's and St. Luke's Hospitals of Duluth during the ten-year period ending December 31, 1952.

The case histories of 424 patients were reviewed (Table I). There were 341 males and 83 females; a ratio of four to one. Approximately 30 per cent of the patients were under forty-five years of age.

The severity of the hemorrhage was arbitrarily graded mild, moderate, and massive (Table II). In mild hemorrhage, the hemoglobin range was from 100 per cent down to 75 per cent, and the red blood cell from five million down to three and one-half million. In moderate hemorrhage, the hemoglobin ranged from 75 per cent down to 50 per cent and the red blood cell count from three and one-half million to two and one-half million. In massive hemorrhage, the hemoglobin ranged from 50 per cent downward and the red blood cell count from two and one-half million on down. Seventy-one cases were graded as mild, 179 as moderate, and 174 as massive hemorrhage (Table III). Patients over forty-five years of age showed

TABLE I. CASES OF UPPER GASTROINTESTINAL HEMORRHAGE REVIEWED

| | |
|---|---|
| Males | 341 |
| Females | 83 |
| Total | 424 |

TABLE II. RANGE OF HEMOGLOBIN AND RED BLOOD CELL COUNT

| | Hbg. | RBC |
|---|---|---|
| Grade 1 Mild | 100—75% | 5 M—3.5 M |
| Grade 2 Moderate | 75—50% | 3.5 M—2.5 M |
| Grade 3 Severe (massive) | 50— 0% | 2.5 M—0 M |

essentially the same distribution in the grades as those under forty-five years of age (Table IV). Ninety-four (22 per cent) of the cases had no record of an x-ray examination and in twenty-three (5 per cent) no abnormality could be demonstrated by x-ray examination or exploratory operation (Table V). In those with an established diagnosis, the bleeding was due to duodenal ulcer in 70 per cent, gastric ulcer in 10 per cent, and a variety of pathologic conditions in the remaining 20 per cent.

A review of these cases also yielded these miscellaneous observations: (1) alcohol and irregular eating habits seemed frequently to predispose to bleeding; (2) esophageal hemorrhage was usually massive and recurrent hemorrhages were common; (3) thirty-seven patients had had forty-two operations mostly for duodenal ulcer or its complications; (4) the x-ray diagnosis of gastritis and gastroduodenitis was fairly common. However, it is possible that in some of these patients the hemorrhage was due to tiny mucosal erosions not detected by x-rays; (5) less common lesions of the stomach, duodenum and jejunum should always be kept in mind. This is especially true of lesions of the jejunum and the second and third portions of the duodenum; (6) fatal hemorrhage occasionally followed reduction of fractures and other unrelated abdominal operations.

Since the treatment of these patients was carried out by many physicians, variations were common. Eighty-one patients were operated upon; seven as emergencies, sixty-six while still in the hospital but after bleeding was controlled, and eight returned to the hospital at a later date for

From the Department of Surgery, Duluth Clinic.

TABLE III. SEVERITY OF HEMORRHAGE

| | |
|---|---|
| Grade 1 (mild) | 71 |
| Grade 2 (moderate) | 179 |
| Grade 3 (severe) | 174 |
| Total | 424 |

TABLE IV. UPPER GASTROINTESTINAL HEMORRHAGE BY AGE

| | | | |
|---|---|---|---|
| Under 45 years | | | |
| Grade | 1 | 2 | 3 |
| Number | 24 (20%) | 49 (41%) | 47 (39%) Total 120 |
| Over 45 years | | | |
| Grade | 1 | 2 | 3 |
| Number | 52 (17%) | 127 (41%) | 125 (41%) Total 304 |

TABLE V. LESIONS DIAGNOSED BY EXAMINATION

| | |
|---|---|
| Duodenal ulcer | 217 |
| Presumed duodenal ulcer (previous surgery) | 5 |
| Presumed duodenal ulcer (spastic duodenum) | 4 |
| Gastric and duodenal ulcer | 6 |
| Gastric ulcer | 34 |
| Carcinoma stomach | 6 |
| Cirrhosis and varices | 5 |
| Stomal ulcers | 7 |
| Hiatal hernia | 3 |
| Banti's disease—Varices | 3 |
| Sarcoma stomach | 1 |
| Leiomyomata jejunum | 2 |
| Fibroma stomach and Meckel's diverticulum | 1 |
| Carcinoma duodenum | 2 |
| Polyp duodenum | 1 |
| Negative examination | 23 |
| No examination | 94 |
| Total | 424 |

TABLE VI. TIME OF SURGERY

| | |
|---|---|
| Emergency operation (2 deaths) | 7 |
| In hospital | 88 |
| Late | 8 |
| Total | 81 |

TABLE VII. LESIONS OPERATED UPON

| | |
|---|---|
| Duodenal ulcer | 53 |
| Gastric ulcer | 6 |
| Duodenal and gastric ulcer | 2 |
| Stomal ulcer | 4 |
| Carcinomatous ulcer | 4 |
| Banti's disease (splenectomy) | 3 |
| Leiomyomas jejunum | 2 |
| Sarcoma stomach | 1 |
| Carcinoma duodenum | 2 |
| Negative | 4 |
| Total | 81 |

TALE VIII. SURGICAL DEATHS (SIX) IN SEVENTY-THREE CASES

| | | |
|---|---|---|
| 1. | M (65) Resection stomach | Blowout duodenal stump. |
| 2. | M (62) Resection stomach | Erosion of pancreatico-duodenal artery |
| 3. | M (33) Resection stomach | Evisceration—non-functioning stomach |
| 4. | M (53) Closure acute perforation resection stomach | Blowout duodenal stump. |
| 5. | M (61) Exploratory | G. I. hemorrhage—4 days postoperative |
| 6. | M (63) Resection stomach | G. I. hemorrhage—18 days postoperative |

TABLE IX. MORTALITY ACCORDING TO TREATMENT

| | |
|---|---|
| Operated upon (Grades 2 and 3) | 65 |
| Deaths | 6 |
| | 9% |
| Autopsies | 5 |
| Treated medically (Grades 2 and 3) | 282 |
| Deaths | 38 |
| | 13.5% |
| Autopsies | 21 |

TABLE X. MORTALITY ACCORDING TO AGE

| | |
|---|---|
| Under age 45 years | |
| Patients (Hemorrhage, Grades 2 and 3) | 96 |
| Deaths | 6 |
| | 6.2% |
| Over age 45 years | |
| Patients (Hemorrhage, Grades 2 and 3) | 252 |
| Deaths | 38 |
| | 15% |

surgery (Table VI). In this group sixty-five had had moderate or massive bleeding. The majority (80 per cent) were found to have benign ulcerations of the duodenum or stomach. In 15 per cent, the lesion belonged to a miscellaneous group, mostly benign or malignant tumors. In 5 per cent, no source of bleeding was found (Table VII).

There were six postoperative deaths, a mortality of 9 per cent. Five followed resection of the stomach for bleeding duodenal ulcer. Of these, two were due to late hemorrhage, the result of erosion of the pancreatico-duodenal artery; two were the result of a blown-out duodenal stump and one patient had a non-functioning stoma and complete wound dehiscence. One death followed negative exploratory operation in an individual who died four days postoperatively from another hemorrhage (Table VIII). Postmortem examination revealed a small ulcer high on the posterior wall near the lesser curvature in the base of which was an open blood vessel, a branch of the left gastric artery.

By comparison, 282 patients with grade II and III hemorrhage were treated medically. In this group, there were thirty-eight deaths, a mortality of 13.5 per cent (Table IX).

There were six (6.2) deaths among ninety-six patients below the age of forty-five with grades II and III hemorrhage, while in the group over forty-five years with grades II and III hemorrhage, there were thirty-eight deaths, a mortality

of 15 per cent (Table X). Fatal exsanguination occurred in one half of the cases in the older group. Also, as one might expect in the older age group, co-existing lesions were common. Diabetes, cirrhosis, hypertension, hypertensive cardiovascular disease, coronary sclerosis, and arteriosclerosis were frequently associated.

## Discussion

A more aggressive surgical attitude is in order because, in the first place, the past decade has shown a remarkable advance in major surgery as evidenced by a mortality rate of less than 2 per cent for partial gastrectomy for peptic ulceration of the stomach or duodenum. Antibiotics, blood banks, and better anesthesia and techniques are among the factors contributing to this improvement. Secondly, medical management has yielded no comparable improvement.

When bleeding continues or recurs despite adequate medical care, surgical intervention is imperative. At operation, the abdomen should be thoroughly explored. The source of bleeding, if found, should be controlled. If it is a duodenal ulcer, the pancreatico-duodenal artery should be ligated, the ulcer excised if possible, and two-thirds or three-fourths of the stomach resected. If the source of bleeding is a gastric ulcer, two-thirds or three-fourths of the stomach should be resected, including the pylorus, the antrum, and the greater part of the lesser curvature. The left gastric vessels should always be ligated and divided. In those patients in which exploration, including duodenotomy and gastrotomy, does not reveal the source of bleeding, it is mandatory to perform a partial gastrectomy and this must again be a two-thirds or three-fourths resection with ligation and division of the left gastric artery.

## Summary

A study of 424 cases of upper gastrointestinal bleeding is presented.

In 80 per cent of the cases, peptic ulceration was the cause of bleeding. In 15 per cent, there were other lesions of the stomach, duodenum, and jejunum. In 5 per cent, routine examinations or exploratory operations failed to disclose the source of bleeding. Sixty-five patients with moderate or severe hemorrhage were operated upon with a mortality of 9 per cent. The cause of death in each case was due to a failure in surgical technique or judgment.

The mortality under medical management was 13.5 per cent.

The death rate for upper gastrointestinal hemorrhage in those below forty-five years of age was 6 per cent as compared to 15 per cent in individuals over forty-five years. Exsanguination was the mode of exit in half of those cases in the older age group.

## Conclusion

A more aggressive surgical attitude towards severe upper gastrointestinal hemorrhage is justified.

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## PSITTACOSIS REPORTED

According to the U. S. Public Health Service, psittacosis virus was recently recovered from two parrots sent to a game farm in Virginia. X-ray examination of the attendant who cared for the birds showed evidence of pneumonitis about one week after discharge from a hospital where he was treated for pneumonia. A specimen of blood from the attendant was positive 1:8 with Lygranum antigen.—Psittacosis infection was reported in three parakeets in Connectciut, one of which had been imported from Florida and two from New York City from a source which had previously been incriminated. Reports have been made of four suspected cases in human beings and of two actual cases of psittacosis, in both of which the blood showed a positive complement fixation test in a titer of 1:128.

Pursuant to information received from the Tennessee health commissioner, indicating that human cases of psittacosis in that state had been traced to contact with parakeets purchased in North Carolina, an investigation by the North Carolina State Board of Health disclosed six cases of atypical pneumonia in the family of the owner of the aviary from which the parakeets had been purchased. It was reported that a large number of persons raise psittacine birds in this area and that many cases of atypical pneumonia have occurred. Blood samples and several birds have been sent to the virus laboratory of the Communicable Disease Center.—J.A.-M.A., Vol. 154, No. 12, March 20, 1954.

# OBSTRUCTION OF THE SMALL INTESTINE

## Survey of Cases in a Small Group Practice

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OBSTRUCTION of the small intestine is an important surgical condition, since almost certain death results if it is left untreated. Special impetus to research problems in obstruction has emanated from the University of Minnesota. Emphasis by Wangensteen on correction of electrolytic and fluid balance and early decompression with or without laparotomy has resulted in a decreased mortality. His complete and concise summary of this subject is readily available.[1]

All of the cases of small bowel obstruction in the past twenty-five years treated only by operative means at the Mankato Clinic are presented. Major surgical procedures at both the Immanuel and St. Joseph's Hospitals totaled 15,740, and of these 257 patients were operated upon for small bowel obstruction. There were fifty-six hospital deaths in this group (Table I).

TABLE I. SMALL BOWEL OBSTRUCTION SURVEY
Mankato Clinic (1927-1952)

| | No. Cases | Percent |
|---|---|---|
| Total major surgical procedures (through 1952) | 15,740 | |
| Small bowel obstruction treated by operative means | 257 | 1.3% |
| Hospital deaths | 56 | 22% (1947-1952) 6.5% |
| Site of Obstruction | | |
| Low | 250 | 97% |
| High | 5 | 2% |
| Unknown | 2 | 1% |
| Type of Obstruction | | |
| Strangulated | 130 | 50+% |
| Simple | 127 | 49+% |

The clinical picture of intermittent crampy abdominal pain, nausea, vomiting, and distention is well known. Most patients with suspected obstruction were treated with indwelling nasal catheter drainage, abdominal hot moist packs, intravenous fluids, and analgesics. The degree of distention was evaluated periodically with abdominal x-rays and simply measuring the circumference of the abdomen with a tape. Failure to respond to this regime in twenty-four hours, as evidenced by increasing toxicity and distention, prompted operation. Patients with an obviously strangulated hernia or suspected strangulated gut were immediately operated upon. If the surgeon's description indicated compromising of the blood supply, the obstruction was noted as being of strangulated type; all others were considered simple. The exact site of the obstruction was difficult to determine. Again, the description by the surgeon of the operative findings was used as a means of classification. Duodenal obstruction was not included since this organ for the most part was obstructed at or near the pylorus by gastric or related lesions. In general, if the location of the obstruction was jejunal, it was classed as high, and if in the lower one-third of the ileum it was considered low. Admittedly, this was not too reliable when dealing with distended bowel of questionable viability, but it served to indicate that most of the lesions were in the lower small bowel (Table I). Multiple surgical procedures were utilized and sometimes several in the same situation. It was our operative policy to facilitate decompression by the easiest means, to remove devitalized tissue and re-establish continuity if possible. Before the more common use of intestinal intubation by an indwelling nasal catheter, ileostomy was an accepted method of combating paralytic ileus. The patients in our study commonly had ruptured appendices or other septic conditions and were in extremely poor condition. The additional trauma of this procedure and the absence of antibiotics particularly, contributed heavily to the over-all mortality. The lack of knowledge regarding principles of treatment now well established and the fact that transfusions were not ordinarily used were also contributory to this high figure in early days. The patients expiring in our hospitals had various causes of death, most of which were quite obvious, though few were proven by autopsy. This is unfortunately common in many private institutions.

The series was equally divided as to sex, the male patients being but slightly more commonly afflicted than female. In this section, there are virtually no colored people so all of the patients were white. As one might surmise, most of the patients were over fifty years of age. Youth and early middle age contributed 24 per cent of the operations, while there were few in the very young (Table II).

TABLE II. SMALL BOWEL OBSTRUCTION SURVEY

| | No. Cases | Percent |
|---|---|---|
| Sex and Color | | |
| Male | 135 | 53% |
| Female | 122 | 47% |
| Black | 0 | — |
| White | 257 | 100% |
| Age | | |
| 50 and above | 153 | 59+% |
| 20-50 years | 62 | 24% |
| 5-20 years | 18 | 7% |
| 1- 5 years | 8 | 3% |
| Below one year | 16 | 6% |
| Duration of Symptoms | | |
| More than 48 hours | 117 | 45+% |
| 24-48 hours | 15 | 6% |
| 12-24 hours | 65 | 25% |
| Less than 12 hours | 51 | 19% |
| Not known | 9 | 3% |

Unfortunately, 45 per cent of the patients had symptoms which lasted more than forty-eight hours. This was a definite contributory factor to mortality as will be shown later. A fair number were operated upon in less than twelve hours. These were mostly strangulated external hernia in which the diagnosis was readily apparent and of necessity came to early operation. Although the site of the obstruction was difficult to determine, there was a strikingly high incidence of obstruction in the lower small bowel. The number of strangulated and simple types were equal (Table II).

The obstructions were caused predominantly by herniations. Forty-six per cent of our cases were due to hernia of one type or another. None was included which did not have an obstructed loop of small bowel. Several such loops reduced themselves after spinal anesthetic was administered. We prefer this type of anesthetic. The surgeon's notes, however, indicated that the bowel showed definite evidence of obstruction and usually strangulation. Both of our cases of internal hernia were due to defects in the omentum of the lower ileum. Postoperative adhesions were found in seventy-two cases. Talc used in preparing rubber gloves has proven to be a peritoneal irritant and causes adhesions. For several years now, as elsewhere, nonirritating starch powders have been substituted by our operating staff. Ruptured appendices obstructed either mechanically due to abscess formation or by adhesions in twenty-four cases. This was found chiefly in the early days of clinic practice. All of the intussusception cases were of the ileocecal type. Ileus, as we know it today, was treated at that time by surgical intravention and in nine cases an appropriate ileostomy was performed. Carcinoma of the bowel in every case orginated in the colon and

TABLE III. ETIOLOGY OF OBSTRUCTION

| | No. Cases | Percent |
|---|---|---|
| Hernia | 119 | 46% |
| Inguinal | 61 | 24% |
| Femoral | 29 | 11% |
| Incisional | 20 | 8% |
| Umbilical | 7 | 3% |
| Internal (omental rent) | 2 | 1% |
| Adhesions | 72 | 28% |
| Ruptured appendix | 24 | 10.5% |
| Intussusception | 11 | 4% |
| Ileus | 9 | 3.5% |
| Carcinoma | 6 | 2% |
| Volvulus | 3 | 1% |
| Gallstone | 3 | 1% |
| Congenital bands | 3 | 1% |
| Mesenteric thrombosis | 2 | Less than 1% |
| Pin perforation | 1 | Less than 1% |
| Abdominal injury (hematoma) | 1 | Less than 1% |
| Pelvic abscess | 2 | Less than 1% |
| Regional illeitus | 1 | Less than 1% |
| Not known | 0 | |

TABLE IV. OPERATIVE PROCEDURE AND MORTALITY

| | No. Cases | Percent |
|---|---|---|
| Operative Procedures | | |
| Release bowel | 128 | 50% |
| Repair hernia | 113 | 44% |
| Release bowel and repair hernia | 108 | 42% |
| Release adhesions | 63 | 24% |
| Ileostomy | 61 | 24% |
| Bowel resection | 15 | 6% |
| Remove stone | 3 | 1% |
| Short circuit | 3 | 1% |
| Remove pin | 1 | Less than 1% |
| Mortality | | |
| Peritonitis | 31 | 55% |
| Shock | 6 | 10% |
| Toxemia | 5 | 9% |
| Carcinomatosis | 4 | 7% |
| Cardiovascular | 4 | 7% |
| Not known | 3 | 5% |
| Pulmonary embolism | 2 | 3% |
| Gas gangrene | 1 | 1% |

caused blockage by peritoneal spread or metastases to the small gut. Other sources, volvulus, gallstones, bands, et cetera, were found very infrequently (Table III).

The operative procedures in the various cases were dependent upon the type and cause of obstruction. Simple lysis of adhesions was accomplished in one-fourth of the cases. Ileostomy was deemed necessary in one-fourth of this group also, either as a proximal decompression safety valve in resections which were necessary in a fair number of instances, or as a direct attack upon the obstruction *per se*. Because of the high incidence of hernia, release of the bowel and repair of the hernia by one method or another was done most often. As one can readily see, circumstances usually permitted this combination, but in a few instances doubtful tissue viability prevented hernia repair (Table IV).

Investigation showed that peritonitis was by all odds the biggest cause of death. It was difficult to separate shock and toxemia in obstructed patients, but in earlier days shock following oper-

TABLE V. SUMMARY OF MORTALITY
56 Cases

| | |
|---|---|
| Sex | |
| Male | 53% |
| Female | 47% |
| Age | |
| Above 50 | 66 % |
| 20-50 | 20.7% |
| 5-20 | 5.7% |
| 1- 5 | 2 % |
| Below 1 | 5.7% |
| Type | |
| Simple | 62% |
| Strangulated | 38% |
| Duration of Symptoms | |
| More than 48 hours | 64% |
| 24-48 hours | 6% |
| 12-24 hours | 26% |
| 12 hours | 3% |

TABLE VI. ETIOLOGY—PROCEDURE—MORTALITY
56 Cases

| | |
|---|---|
| Etiology | |
| Adhesions | 25% |
| Ruptured appendix | 25% |
| Hernia | 20% |
| Inguinal | 9% |
| Incisional | 9% |
| Femoral | 2% |
| Ileus | 7% |
| Carcinoma | 6% |
| Intussusception | 5% |
| Mesenteric thrombosis | 5% |
| Volvulus | Less than 2% |
| Gallstone | Less than 2% |
| Pin | Less than 2% |
| Congenital band | Less than 2% |
| Internal hernia | Less than 2% |
| Procedure | |
| Ileostomy | 58% |
| Release bowel | 22% |
| Repair hernia | 18% |
| Release bowel and repair hernia | 17% |
| Release adhesions | 15% |
| Bowel resection | 9% |
| Remove gallstone | 3% |
| Remove pin | Less than 2% |
| Incision and drainage | Less than 2% |

ation was common, and that is what this figure represents. Carcinomatosis due to primary carcinoma of the colon in our series was found to be the cause of four deaths. Cardiovascular disturbances, coronary occlusion, and pulmonary embolism were found in a surprisingly small number of cases, considering the high incidence of our patients in the upper age groups (Table IV).

In summarizing our mortality, male and female patients were involved practically identically. Sixty-six per cent of the dead patients were over fifty years of age. Curiously enough, simple obstruction was found in 62 per cent of the fatal cases, while strangulated conditions existed in only 38 per cent. As previously mentioned, the duration of symptoms was a definite factor in mortality, 64 per cent of our patients having symptoms more than two days (Table V). Adhesions were found in 25 per cent and hernias in 20 per cent. The remainder of the etiological agents were of lesser significance. Ileostomy was done in 58 per cent of the patients who died, which would indicate that if such a procedure were deemed necessary, the patient was in very critical condition or tissue viability prevented reestablishment of continuity. It did not mean presumably that the operation itself was a contributing factor in the death, although, in earlier days, the additional insult of such a procedure was a definite factor in the high mortality which we have encountered (Table VI).

It is interesting to note that a male or female patient with small bowel obstruction had a one in five chances of death. In those fifty years of age or above and, curiously enough those with a simple hernia rather than a strangulated hernia, chances of survival were considerably decreased. All of our patients with mesenteric thrombosis expired. Internal hernias, likewise, carried a high mortality. Half of the patients requiring ileostomy expired.

During the past five years, with strict attention to the excellent principles mentioned and earlier operation in questionably secure patients being treated conservatively, our mortality has dropped to 6.5 per cent (Table I).

### Reference

1. Wangensteen, Owen: Intestinal obstruction. Ed. 2. Chaps. 5 and 6. Springfield, Illinois: Charles C Thomas Co., 1942.

## THE FAMILY DOCTOR

The family doctor has in general a commendable composite of wide interests or receptivity to ever-broadening developments in the various aspects of total medical practice, of an inevitable understanding of his patients that goes beyond Johnnie's pimples and Mary's menstrual cramps and Susie's loss of weight due to lack of appetite—beyond the peptic and other complaints and into life stresses and global biologic considerations. These aspects of medical practice on the part of the family doctor are inevitable because he finds himself as a matter of course in the peculiar position of friend, adviser, and dependency symbol; this is the complete physician, this is the family doctor.—Jess V. Cohn, M.D., in *Pennsylvania Medical Journal.*

# THROMBOTIC THROMBOCYTOPENIC PURPURA

## Report of a Case

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THROMBOTIC thrombocytopenic purpura is characterized pathologically by generalized hyaline arteriolar and capillary thrombi and clinically by a fulminating thrombocytopenic purpura accompanied by hemolytic anemia and transient renal, cardiac, and neurological manifestations. It was first reported as a newly recognized disease by Moschcowitz[19] in 1925. The next report of the condition appeared in 1936 when Baeher, Klemperer, and Schifrin[2] clearly defined the syndrome and presented four additional cases. It has been described with increasing frequency in American medical literature during the past five years. An excellent review of the reported cases was made in 1952 by Barondess[3] and he added three new cases of his own. At present, the literature contains descriptions of about forty cases,[1-24] in only a small percentage of which was the diagnosis even considered antemortem. In 1952, Cooper, Stickney, Pease, and Bennett[10] reported two cases in which the diagnosis was confirmed antemortem by examination of aspirated bits of bone marrow. In most instances, the disease has occurred in young adult white women, although it has also been reported in men and in colored people.

The onset of illness is usually abrupt, although occasionally there has been reported a preceding period of vague ill health. The symptoms include malaise, weakness, fever, and frequently suggestions of an upper respiratory infection. Often there have been abdominal pains, anorexia, nausea, and vomiting. Anemia may be present at the onset or may develop during the course of the illness and is usually independent of the bleeding which accompanies the thrombocytopenic purpura. Transfusions have been of little benefit. Frequently jaundice and hyperbilirubinemia have accompanied the condition. Purpura and other hemorrhagic manifestations, such as melena, hematemesis, and hematuria, invariably have eventually been part of the picture. Aside from the hemorrhagic manifestations of the thrombocytopenic purpura, transient and bizarre neurological disturbances of almost any type have been distinctive clinical features. There may be headache, dizziness, delirium, confusion, drowsiness, and progressing coma. Transient paralysis or paresis are very common. The disease, in all reported cases, has been fatal, usually in from two to four weeks, varying from one week to three months. Engle[12] reported a case which had a partial remission of about one month's duration, but this is extremely rare.

In thrombotic thrombocytopenic purpura, the blood shows the typical manifestations of a hemolytic anemia, although the severity of the anemia and the rate of the hemolysis have varied greatly. The erythrocyte count and hemoglobin have dropped simultaneously, resulting in a normocytic, normochromic type of anemia. The mechanism of the hemolytic process has been carefully studied by Singer.[21,22] In the cases where the osmotic fragility has been reported, it was normal or increased. There has been no evidence of autoimmunization, transfused cells being destroyed as rapidly as the patient's own cells. The Coomb's test findings, when reported, have been negative. Reticulocytes have increased, and in many cases abnormal young erythrocytes of the normoblastic series have appeared in considerable numbers in the peripheral blood. Bone marrow in most patients in whom it has been studied has shown a moderate hyperplasia with normal megakariocytic activity. There has been, with rare exceptions, a uniformly persistent thrombocytopenia, accompanied by hemorrhagic phenomenon, and prolonged bleeding and clotting times.

The etiology of this condition remains as great a mystery today as it was to Moschcowitz in 1925. The possibility of an allergic reaction has been suggested,[15] but there is little evidence to support this theory. It has also been suggested that the condition may be a primary subendothelial, vascular lesion with secondary accumulation of platelets on the injured endothelial area.[15] The idea has also been advanced that this illness belongs to the so-called collagen group of diseases.

The hyaline thrombi have been described in

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almost every organ but, as Clough[9] pointed out, there is a surprising lack of information regarding the occurrence of lesions in the skin and skeletal muscles.

Treatment thus far has been useless. All cases have terminated fatally. Although there is no apparent evidence of hypersplenism in these cases, splenectomies were done in several instances and were ineffective. Final evaluation of the effect of splenectomy cannot be made at this time, however, particularly since most of the patients on whom the operations were performed were terminally ill at the time the surgery was done.[18] The results of treatment with ACTH, Cortisone, and various antibiotics have been unimpressive. Since there is no evidence, as mentioned above, of hyperimmune bodies, we should not expect benefit from ACTH and Cortisone as pointed out by Hagen.[17]

## Report of Case

A forty-one-year-old white housewife was admitted to the Northwestern Hospital on October 9, 1952, primarily fearing that she was having a stroke.

For six months prior to admission, she had been working and living with painting and staining projects in her home. She had been very active in the painting work, handling paints, stains, and solvents. For about three months before the onset of acute symptoms, she had complained of weakness and exhaustion out of proportion to the amount of work she did. For a month prior to admission, she had been suffering from severe frontal headaches to which she had previously not been subject. She suffered from aches and pains in her legs. Because of these complaints, she had gone to a rest hospital for five days "to get a good rest away from her family." On October 8 her hemoglobin was 45 per cent. On the night of admission to Northwestern Hospital, she had experienced a transient aphasia and right-sided paresis.

Review of her past history revealed that the patient had had the usual childhood diseases without complication. She had always enjoyed good health and had been a very active, vigorous woman. She had an appendectomy in 1926, a tonsillectomy in 1946, and plastic surgery on the fallopian tubes in 1938. Menses started at age thirteen, and occurred every twenty-seven to thirty-five days with a normal flow for six days. Her systemic history was entirely negative and her routine medical examinations had always been reported as negative. When she was examined on March 11, 1952, there were no abnormal findings. At that time urinalysis was negative. The hemoglobin was 12.3 grams; erythrocyte count, 4,240,000; leukocyte count, 9,800 with 78 per cent neutrophils, 20 per cent lymphocytes, 1 per cent eosinophils, and 1 per cent monocytes. Erythrocyte sedimentation rate was 20 mm. in one hour. Stool was negative for occult blood.

Physical examination on admission to Northwestern Hospital revealed a well-developed, fairly well-nourished, white woman who appeared to be very apprehensive. Her skin and mucous membrane were pale. The eyes, including the fundi, were normal. The ears, nose, and throat were within normal limits. The tongue was normal. Examination of the neck revealed no adenopathy and no limitation of motion. Examination of the chest revealed no abnormality. The heart tones were normal; the cardiac rate was 100 per minute with a regular sinus rhythm. Examination of the abdomen revealed no palpable masses or viscera. There was no tenderness or rigidity. The abdomen was soft. Neurological examination was entirely negative. Blood pressure was 120/80. There were small hematomas on the shins of both legs. There were no petechiae noted.

On admission, the electrocardiogram was normal. The hemoglobin was 5.7 grams; erythrocyte count, 1,590,000, and the leukocyte count, 10,000 per cu. mm.

Shortly after admission, the patient complained of precordial pain. Because of this pain and low hemoglobin, she was given 1000 cc. of whole citrated blood intravenously during the first twelve hours following admission. On October 10, 1952, a bone marrow biopsy was done. Study of the bone marrow revealed a hematocrit of .5 per cent fat, 66.5 per cent plasma, 1 per cent myeloid-erythroid layer, 32 per cent erythrocytes. Study of the smears from the myeloid-erythroid layers showed a marked increase in the developing red cells. These were normoblastic cells. A mild increase in the plasma cells was also present and a normal number of megakariocytes was noted. There was no evidence of leukemia, tumor, or granulomata.

Further laboratory studies on October 10, 1952, revealed a hemoglobin of 8.4 grams; erythrocyte count, 2,400,000, and leukocyte count, 7,300. Differential study of the white blood cells revealed 73 per cent neutrophils, 24 per cent lymphocytes, 1 per cent monocytes, and 2 per cent eosinophils. The reticulocyte count was 14 per cent. The platelet count was 39,000. A urinalysis was normal. The bleeding time was 7 minutes 45 seconds; clotting time, 3 minutes. The prothrombin time was normal. The erythrocyte sedimentation rate was 27 mm. in 60 minutes.

Further laboratory studies within a few days after admission revealed normal blood urea nitrogen, normal cephalin cholesterol flocculation test, normal thymol turbidity test, no occult blood in the stool, and free hydrochloric acid in the gastric analysis. Coomb's test findings were negative on two occasions.

X-ray examination showed a normal chest, normal esophagus, stomach, and duodenum, following the ingestion of a barium meal, and a normal colon except for marked redundancy throughout. Repeated electrocardiograms showed no change and were essentially the same as those made in March, 1952, and February, 1951.

In spite of repeated transfusions (a total of 6,000 cc. of blood) during her hospital stay, the hemoglobin and erythrocyte count remained low. The platelet count varied between 18,000 and 52,000. The patient's red blood cell osmotic fragility was tested. Her cells began to hemolize at .54, and hemolysis was complete at .32.

Fig. 1. Heart. Note the multiple petechial hemorrhages on the endocardial surface.

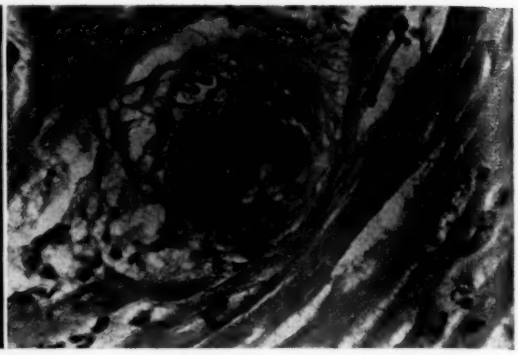

Fig. 2. Heart muscle with a typical lesion. Note the hyaline thrombus in the arteriole.

Control cells began at .46 and were complete at .34. There was no evidence of spherocytosis in any of the blood smear studies. The serum bilirubin was slightly elevated to 1.3 mg. Of this total, 0.12 mg. was direct acting. The thymol turbidity test showed 1 unit. Cephalin cholesterol flocculation test revealed one plus in twenty-four hours and 2 plus in forty-eight hours. On October 13, 1952, the quantitative urine urobilinogen was .9 units per 100 cc. of urine. Examination of the gastric contents on October 14, 1952, revealed normal free hydrochloric acid of 50 degrees and a total acid of 59 degrees.

On October 17, 1952, purpura was first noted on the patient's legs. On October 20, the Guaiac test on the stool was positive for the first time. On October 20, 1952, the possibility of thrombotic thrombocytopenic purpura was considered in the differential diagnosis. At this time Cortone was started by mouth, 25 milligrams four times a day.

On October 22, 1952, the patient started to menstruate with excessive blood loss. She also started to have bloody emesis at this time. Her liver became very tender and palpable at the costal margin. At times it was thought that the spleen could also be palpated in the left upper quadrant.

On October 25, 1952, the patient had repeated bloody emeses and was unable to keep the Cortone down. This drug was discontinued and ACTH, 100 mg., was given at once and 50 mg. every eight hours thereafter, intramuscularly.

During the evening of October 25, 1952, the patient was seen in consultation with Dr. C. J. Watson, who concurred in our diagnosis of hemolytic anemia and thrombocytopenic purpura and also with the possibility that this might be a thrombotic thrombocytopenic purpura. On October 27, 1952, the bone marrow slides were examined by Dr. Bennett and Dr. Stickney of the Mayo Clinic. While there were no thrombi visible in the sections we examined, Dr. Bennett thought there were some suggestive changes in some of the vessels, and suggested further bone marrow studies be made in an attempt to find the diagnostic lesions. The patient expired suddenly on October 28, 1952, nineteen days after admission to the hospital. During the nineteen days her temperature ranged from 98 to 100 degrees.

*Autopsy* (four hours after death) was performed and described by Dr. Frederick H. Lott.

General Appearance: The body was that of an adult white woman, measuring 158 cm. in length. Estimated weight was 125 pounds. The body was well developed and well nourished. Postmortem rigidity was present, and there was a mild postmortem lividity in the dependent parts. There was no edema or jaundice. The pupils were sound and equal. The ears and nose showed nothing of note. The teeth were in a good state of repair. The external genitalia showed evidence of bleeding, and the anus showed nothing of note. There were numerous small petechial hemorrhages over much of the body surface, many of which were secondary to injection. There was a well-healed lower abdominal scar which measured 16 cm. in length. There was a well-healed McBurney's scar measuring 12 cm. in length.

Abdominal and Thoracic Cavities: On opening the abdomen, the subcutaneous fat was found to measure 2 cm. in thickness. The peritoneal surfaces were smooth and shiny, and there was no excess fluid present. The appendix had been surgically removed. The liver extended 10 cm. below the xiphoid process and 7 cm. below the costal margin in the right midclavicular line. The diaphragm arched to the fourth rib on the right, and to the fourth intercostal space on the left. On opening the thorax, the lungs were found to be free and the pleural cavities empty.

Heart: The pericardial sac showed nothing of note save for a slight excess of clear amber fluid. The heart measured 13 cm. in width. Examination of the larger branches of the pulmonary artery showed no evidence of thrombi or emboli. The heart weighed 310 grams. The valves were normal in appearance. Examination of the coronary arteries showed nothing of note. The myocardium was somewhat flabby in consistency, and on cut section had a markedly mottled appearance, with small petechial hemorrhages separated by small greyish-yellow areas, which suggested fibrosis. This was extremely marked in both ventricles and in the interven-

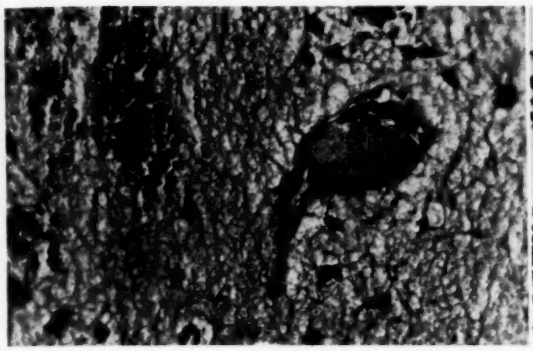

Fig. 3. Brain. Showing typical hyaline thrombi in the arteriole.

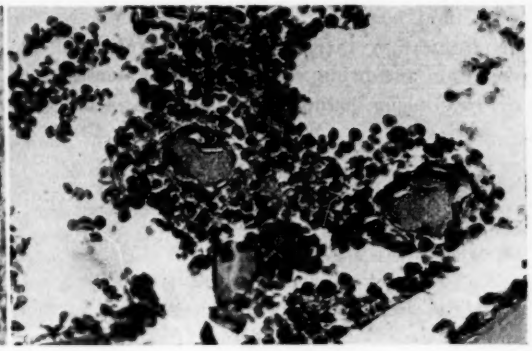

Fig. 4. Bone marrow. Showing two arterioles occluded by hyaline thrombi. This specimen was obtained at autopsy. The antemortem specimens unfortunately failed to show the lesions.

tricular septum. The petechial hemorrhages showed on the endocardial surface of both the right and left ventricles. The root of the aorta was free from atheromatous degeneration. The auricular appendages contained no thrombi.

Lungs: The right lung weighed 200 grams. The surface of the organ was smooth and crepitation was slightly reduced throughout. On cut section, the cut surface showed a very slight excess of blood and fluid, but there was no evidence of consolidation. The left lung weighed 180 grams. The surface of the organ was smooth, and crepitation was slightly reduced throughout. On cut section, the cut surface showed a slight excess of blood and fluid, but there was no evidence of consolidation.

Liver and Gallbladder: The liver weighed 1,900 grams. The surface of the organ was smooth and the edges sharp. On cut section, the cut edge everted, and the cut surfaces showed the markings to be indistinct. There was no gross evidence of darkening of the centers of the lobules. The gallbladder wall was normal in thickness. No stones were present. The common bile duct was patent.

Spleen: The spleen weighed 175 grams. The capsule wrinkled easily. On cut section, the edge everted and the cut surface was red in color. The pulp was soft in consistency. The splenic corpuscles were barely visible.

Pancreas and Adrenals: The pancreas and adrenals showed nothing of note.

Kidneys: The right kidney weighed 150 grams. The capsule stripped easily, leaving a smooth surface. On cut section, the cut edge everted, and the cut surfaces showed the markings to be indistinct. There was no evidence of atrophy of the cortex. The pelvis and ureters showed nothing of note. The left kidney weighed 165 grams. It showed a picture similar to that of the opposite organ.

Bladder and Genital Organs: The bladder contained a few cubic centimeters of a clear yellow fluid, but the bladder mucosa showed numerous hemorrhagic areas. The uterus was normal in size, and, on cut section, contained a small amount of clotted blood. The endometrium was smooth and the cervix showed nothing of note. The fallopian tubes were free and the fimbriated ends appeared patent. On cut section, they were normal in caliber. The ovaries were normal in size, and, on cut section, showed nothing of note.

Abdominal Aorta: The abdominal aorta showed a very mild degree of atheromatous degeneration.

Gastrointestinal Tract: The gastrointestinal tract showed numerous petechial hemorrhages.

Head: On opening the head, the scalp and calvarium were found to be normal in appearance. Examination of the brain showed approximately twenty petechial hemorrhages measuring up to 2 mm. in diameter in the cortex of the brain. These were most pronounced on the right side. The entire brain substance was slightly softened, but otherwise there was no evidence of gross hemorrhages, tumor, or softening. The pituitary gland was normal in appearance.

Microscopic examination showed innumerable hyaline thrombi within the capillaries and arterioles of all the major organs including the bone marrow. These were especially numerous in the myocardium and brain. Associated with these thrombi were scattered small hemorrhages which were surrounded by beginning degeneration of the myocardium and brain. The balance of the sections showed nothing of note.

*Diagnoses:* Thrombotic thrombocytopenic purpura, with petechial hemorrhages in the heart and brain.

## Discussion

While this disease is undoubtedly rare, it is probably more common than the number of cases which have been reported since 1936 would lead one to believe. Diagnosis is probably missed because of our unawareness of the syndrome. We feel that we are justified in reporting this case in order to bring the syndrome to the attention of a greater number of physicians, pointing out again the salient diagnostic features: transitory bizarre neurological disturbance, thrombocytopenic pur-

pura, and hemolytic anemia. Awareness of the syndrome may facilitate earlier diagnosis of the condition and bring about a better understanding of the etiology, pathogenesis, and treatment.

### Summary

1. A case of thrombotic thrombocytopenic purpura with clinical and pathological findings is reported.

2. The clinical picture is manifested by a fulminating thrombocytopenia, hemolytic anemia, and transient focal neurological abnormalities.

3. The postmortem examination is characterized by widespread hyaline thrombotic lesions involving the arterioles and capillaries. The exact nature of these thrombi has not been determined.

4. Its etiology is unknown, and to date all reported cases have been fatal.

5. The importance of antemortem studies has been pointed out.

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## MALNUTRITION

It is well known that hookworm disease, malaria, and tuberculosis are intimately related to malnutrition in many persons who suffer from these diseases, but whether or not the relationship is causal defies affirmation except in certain individual situations. The sufferer of malaria or hookworm disease may be malnourished because of anemia that reduces his productive and earning capacity and therefore his ability to provide for his food needs; or the malnourished person, as a result of diminished resistance, may fall an easy victim to such diseases. Whatever may be the relationship, it is clear that the well-being of the individual is compromised by malnutrition as well as by certain specific diseases.—Institute of Inter-American Affairs, Public Health Reports, November, 1953.

# HYPERSPLENISM: LEUKOPENIC-THROMBOPENIC SYNDROME

## Report of Two Cases

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LEUKOPENIA with splenomegaly usually presents a troublesome diagnostic and therapeutic problem. In 1924, Felty[4] described the syndrome of chronic arthritis associated with splenomegaly and leukopenia. In 1932, Hanrahan and Miller[5] reported the first case of splenectomy for Felty's syndrome. In 1939, Wiseman and Doan[10] described a syndrome which they called "primary splenic neutropenia." This syndrome was characterized by severe neutropenia of the peripheral blood, splenomegaly, and hyperplasia of the myeloid elements of the bone marrow. The mechanism by which these pathological changes take place has been and still is a much disputed point. One school of thought holds that the cells are removed by abnormal phagocytosis by the hypertrophied reticulo-endothelial cells of the spleen.[3,11] Another school contends that the condition is due to an unusual inhibitory effect on the bone marrow of a hormonal substance secreted by an overactive spleen.[2] Both groups agree that these interpretations are at the present time highly theoretical. In 1946, Doan and Wright reported hypersplenism secondary to other chronic diseases of the spleen.[3] These cases show the same clinical picture as those described in the earlier paper as primary splenic neutropenia by these investigators. A survey of the available literature revealed many cases which fit the primary and secondary syndrome. In 1948, Smith and McCabe[8] and in 1950, Rogers and Langley[6] reported examples with hypoplasia of the bone marrow with complete recovery of hematopoiesis following splenectomy. Since Doan and Wright's publication, several cases have been reported which may be classified as "secondary hypersplenism," which apparently benefited from splenectomy. The problem then becomes: Is there actually a primary splenic neutropenia or is the syndrome a symptom of a disease process in which the etiological factor is not recognized?

It is the purpose of this paper to report two cases of apparent hypersplenism, one of which presents an unusual bone marrow morphology which we believe is unique and instructive, and the other demonstrates a portal hypertension with secondary hypersplenism.

## Case Reports

*Case 1.*—A white woman, aged fifty-four years of age, was referred to the authors by Dr. H. Kath of Wood Lake, Minnesota. Her principal complaints were weakness, fatigue, 42-pound weight loss, and repeated bouts of chills and fever over an eight months' period.

Physical examination showed a thin white woman who appeared to be listless and chronically ill. Her temperature was 100.6°, the pulse 102, and the blood pressure 122/82. The spleen was palpable 6 cm. below the left costal margin. The liver was not palpable.

Laboratory studies showed a leukopenia and neutropenia. The range of the leukopenia is shown in Graph A. Examination of the peripheral blood smear showed 6 per cent immature cells, which were interpreted by various pathologists as atypical immature myeloid cells of the type found in leukemia. Although there was no clinical evidence of leukemia, a bone marrow study done on May 23, 1947, was characteristic of a myeloid leukemia. Marrow preparations and the peripheral blood smears, obtained on June 1, 1947, were then sent to two hematologists, Dr. Hal Downey, University of Minnesota, and Dr. E. M. Schleicher, Minneapolis General Hospital, who independently interpreted the immature cells in the peripheral circulation as non-malignant reticulo-plasma cells and the myeloid hyperplasia of the marrow as myeloid maturation arrest rather than myeloid leukemia. Figure 1 shows a reticulo-plasma cell in the peripheral blood. In addition to the myeloid hyperplasia, the marrow sections showed several epithelioid granulomas, an example of which is shown in Figure 2. Plasma cells and eosinophils formed a loose cuff about the epithelioid structures. No giant cells were seen. We have not been able to find a paper which reports such a lesion in the bone marrow in a case of hypersplenism. This granulomatous lesion was somewhat similar to those described by Schleicher in herpes zoster.[7] Demonstrations of these granulomatous lesions led us to believe that we were dealing with a virus infection superimposed upon or causing the hypersplenism.

On July 10, 1947, in spite of our patient's serious condition and the presence of high fever, it was decided that a splenectomy should be carried out. An 800 gram spleen was removed by Drs. O. J. Campbell and N. C. Plimpton. Microscopic examination of many spleen sections revealed a rather severe reticulum hyperplasia, although no proliferation of malignant cells was seen. Many sinusoids were open and somewhat congested with red blood cells and contained from a few to many reticulo-plasma cells morphologically identical to those seen in the peripheral blood. Since only a few of these reticulo-plasma cells were seen in the bone marrow preparation, it is believed that the majority of cells were proliferated in the spleen.

Bone marrow obtained one month after the splenectomy

Fig. 1. Blood smear showing a reticulo-plasma cell. Six per cent of the leukocytes were this type of cell.

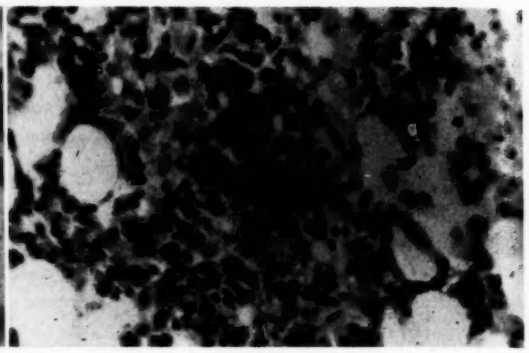


Fig. 2. Epithelioid granuloma in the bone marrow preparation.

showed none of the granulomas previously described, and normal maturation of the myeloid cells was found. Bone marrow aspiration on April 4, 1949, two years after the surgery, showed a normal marrow. The patient made an uneventful recovery and six years postoperatively is enjoying good health. We feel that this patient had hypersplenism without thrombocytopenia. The hypothesis may be advanced that the leukopenia which was corrected following splenectomy may have been in part the result of a chronic infection. We believe that this case points out the necessity of examining sections of the bone marrow as well as smears in cases of hypersplenism for conclusive interpretation of the character of myeloid hyperplasia.

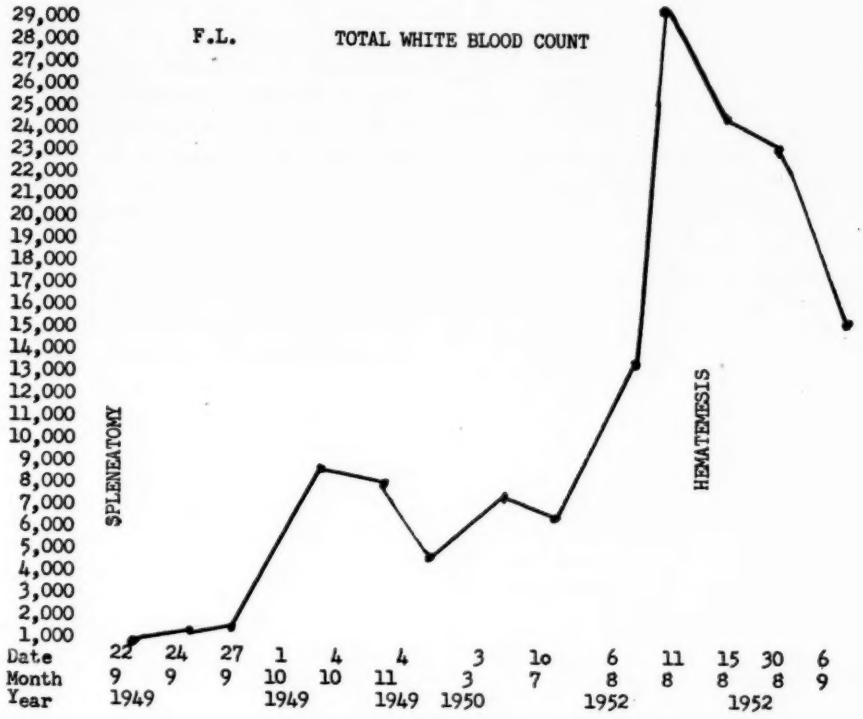
*Case 2.*—A fourteen-year-old boy was admitted to the Northwestern Hospital on December 23, 1949, referred by Dr. E. A. Thayer and Dr. O. E. Wandke of Fairmont, Minnesota. The patient's present illness dated back to September 14, 1949, at which time he had fractured his left forearm while playing football. The fracture had been reduced satisfactorily and the patient returned to his home. On September 17, 1949, he had a hematemesis, losing approximately one quart of blood. He was hospitalized and given two blood transfusions. It was then discovered that he had a marked leukopenia and a splenomegaly. A review of the patient's past history revealed that he had the usual childhood diseases without complications. He had a fracture of the left forearm at age four which healed uneventfully. In October, 1948, he had had a hematemesis but otherwise had enjoyed perfect health. He had been an active child and since entering high school had taken part in athletics.

Physical examination on admission to the hospital revealed a well-developed, fourteen-year-old boy who was pale but otherwise did not appear to be in any distress. Examination of the abdomen revealed the spleen to be palpable 5 inches below the umbilicus. The spleen was hard but not tender. There were no further remarkable physical findings. The left arm was in a well-fitting plaster cast. Laboratory studies on admission to the hospital revealed the patient had a hemoglobin of 7.6 grams; an erythrocyte count of 2,560,000; a leukocyte count of 950 with 31 per cent neutrophils, 64 per cent lymphocytes, 3 per cent monocytes, and 2 per cent eosinophils. His reticulocyte count was 4.6 per cent; the platelet count was 10,000. The bleeding time was 3 minutes and 50 seconds; the clotting time, 5 minutes and 30 seconds. His sedimentation rate was 22 mm. in one hour. The stool was positive for occult blood. The total serum protein was 5.3 grams with 3.4 grams of albumin and 1.9 grams of globulin. The serum bilirubin level was 1.4 mg. per cent. The urinalysis was negative. Bone marrow was obtained by sternal aspiration on September 23, 1949; and smears, imprints, and histological sections were prepared. The preparations showed a marked myeloid hyperplasia with maturation arrest.

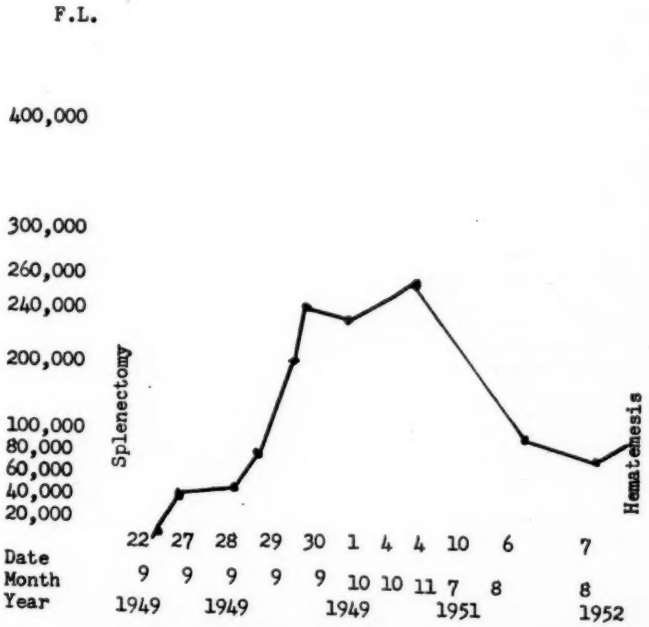
On September 28, 1949, a 1,247 gram spleen was removed by Dr. M. C. Nelson. On gross examination, the spleen was not considered remarkable. The microscopic examination disclosed the sinusoids to be small and not congested. An insignificant amount of fibrosis was present. The lymphoid follicles were relatively small and widely spaced. There was an occasional hemorrhagic area ranging from one to two mm. in diameter. There was nothing seen to suggest a malignant lymphoblastoma or any specific disease.

Postoperatively, the patient's course was uneventful. His hematological response is shown in Graphs 1 and 2. On October 4, 1949, his hemoglobin was 16.8 grams. His erythrocyte count was 5,290,000. His leukocyte count was 8300 with 74 per cent neutrophils, 23 per cent lymphocytes, and 1 per cent monocytes. The platelet count was 232,000. His urine remained normal. The patient was seen again on November 4, 1949; and physical examination was entirely negative. On March 3, 1950, he was seen again, at which time his hemoglobin was 15.6 grams; red blood cell count was 4,560,000; white blood cell count was 8000; and the red blood cell sedimentation rate was 14 mm. in one hour. Differential studies of the blood at this time showed 23 per cent neutrophils, 76 per cent lymphocytes, and 1 per cent eosinophils.

From March 3, 1950, until August 4, 1952, this young man was well and was working as a truck driver. At 11:30 P.M. on August 4, 1952, he was awakened by an emesis of a large quantity of blood. He was taken to the local hospital, where he was given blood transfusions; and then because the hematemesis recurred he was




Graph 1.




Graph 2.

brought to the Northwestern Hospital at 3 A.M. on August 6, 1952. Physical examination on admission to the hospital was entirely negative. The hematemesis continued, necessitating repeated blood transfusions. Eighteen hours after admission, x-ray examination following a barium meal revealed a marked degree of varices extending from the aortic arch to the distal end of the esophagus. Laboratory studies revealed an anemia but no other diagnostic findings. Liver function tests and bone marrow studies were normal.

The hematemesis persisted in spite of balloon tamponage; so, although the patient's condition was poor, an attempt to do a venous shunt to relieve the portal hypertension was made on August 16, 1952. The pressure was measured in a large vessel in the omentum and found to be 385 mm. of blood. The veins in the omentum and on the peritoneum were large and tortuous. The esophagogastric junction was exposed and all the veins from the diaphragm and about the junction were clamped and ligated. The esophagus was pulled down about 4 cm. and packed around its entire circumference with gelfoam. The patient's condition was such that further exploration and shunting procedures were deemed unwise. A liver biopsy was taken and reported as normal.

On August 30, 1952, laparotomy was again undertaken with the plan in mind to establish a portacaval or a splenorenal shunt. The splenic vein was exposed by Blakemore's technique[1] and found to be completely thrombosed, as was the portal vein. In place of the portal vein were a few collateral venus channels which were small and very friable. None of these veins was considered suitable for a shunting procedure.

Following this second operation the hematemesis and melena subsided, and the patient was discharged by ambulance to the hospital in his home town on September 6, 1952, one month after admission on August 6, 1952. From August 4, 1952, to September 6, 1952, he received a total of 71 pints of blood. His hemoglobin varied from 5.3 grams to 16.8 grams and was 13.4 grams at the time of discharge. Leukocyte counts, differential studies, and platelet counts were all normal. After September, 1952, he had three massive gastrointestinal hemorrhages, each necessitating several transfusions. He had a massive hemorrhage in June, 1953, and expired. An autopsy was refused.

It was our original impression that this patient had a hypersplenism with leukopenia and thrombocytopenia preceding his first hematemesis in October, 1948. Now we wonder if this hematemesis and the splenomegaly may have been due to portal hypertension, and whether the portal hypertension should not have been relieved at the time of the original operation.

## Summary

Two cases of hypersplenism are presented—one patient had anemia and neutropenia and the other anemia, neutropenia, and thrombocytopenia. Case 1 had granulomatous lesions in the bone marrow, suggesting a secondary type of hypersplenism. Case 2 simulated a primary portal hypertension with secondary hypersplenism. This second case demonstrated the value of the warning made by Blakemore and Fitzpatrick[1] and earlier by A. O. Whipple,[9] that when a splenectomy is attempted the surgeon should always look for portal hypertension and, if possible, determine its cause. He should be prepared to do a splenorenal or a portacaval shunt if it is indicated. These cases lead us to speculate that perhaps, as more information accumulates, all the cases of so-called primary hypersplenism will prove to be secondary to some other primary condition.

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*The most important elements in the diagnosis of cancer remain those natural attributes of the physician—he must see, hear, and feel the manifestations of malignant processes before utilizing his store of complex tools and methods.*

# FLUID REGULATION IN NEPHRITIS AND NEPHROSIS OF CHILDHOOD

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THE KIDNEY constitutes the main organ for the regulation of fluids and electrolytes. By an active process of filtration, absorption and excretion the kidney defends the normal chemical pattern of the plasma. The maintenance of body stores of water, the acid-base ratio of the blood and tissue cells and the elimination of products of metabolism cannot continue indefinitely in the face of chronic renal disease.

All efforts in fluid and electrolyte therapy for renal disease are based on the assumption that renal function is temporarily or partially inadequate. There is, as yet, no artificial means to maintain indefinitely, an individual with progressive renal failure, recent developments in the artificial kidney notwithstanding.

The efforts of Gamble[4,5] to acquaint members of the medical profession with the fundamental concepts of fluid and electrolyte metabolism are outstanding among those of many researchers in this field. I shall draw upon his contributions in this consideration of the requirements of children with renal disease. What is written here will be confined to a few physiologic aspects of fluid and electrolyte metabolism, on which can be based a rational course of treatment as regards administration of salt and water.

The glomerulus, by a process of filtration, produces a protein-free ultrafiltrate of plasma. The volume of filtrate produced daily approximates 180 liters. Roughly 25 per cent of the cardiac output passes through the glomeruli at any given time. It is generally agreed that the rate of filtration by the glomeruli is relatively constant.

Urea is one of the most important substances removed from the plasma by glomerular activity. Glomerular filtrate has the same acid-base ratio as plasma; that is, a pH of 7.4.

As glomerular filtrate courses past the cells of the renal tubules all but about 2 liters are reabsorbed. In the renal tubules the pH changes from 7.4 to acidity or alkalinity as dictated by the state of metabolic activity and renal function. The limit for acidity of the urine is a pH of 4.8 while the limit of alkalinity of urine is a pH of 7.8.

Two mechanisms for acidification of the urine and conservation of base are employed in normal renal function. The first of these functions consists in substitution of a hydrogen ion supplied by the renal tubule cell for a sodium or potassium ion in the tubular fluid. Glomerular filtrate contains a phosphate with two sodium ions. By substituting a hydrogen ion for a sodium ion half of the base is conserved. The monosodium phosphate is an acid salt and thus tubular fluid is acidified. The degree of acidification may be determined by titration with tenth normal sodium hydroxide to a pH of plasma. The resultant value is usually referred to as "titratable acidity." Above a pH of 7.4, therefore, there would be no titratable acidity, by definition.

A second mechanism for economy of base involves the production of ammonium by tubule cells. The operation of these two adjustments effectively compensates for the excretion of large amounts of acid substances without the depletion of body stores of base. Figures 1 and 2 show quantitatively the adjustment of the production of ammonium and titratable acidity to cover the excretion of acid radicals in the urine.

It is possible to foresee the consequences of chronic renal disease if consideration is given the aforementioned facts. Damage to the glomeruli results in failure to excrete urea, phosphates, sulfates and organic acids. The plasma therefore has a relative deficiency of base; a state of azotemia and acidosis is the consequence. When the renal tubular function is impaired the tubules cannot continue to conserve base and then it becomes necessary for larger amounts to be excreted to maintain the reaction of the urine within the physiologic range. The renal tubules also lose the ability to reabsorb water and stores of body water are rapidly lost.

There is yet another feature of tubular failure which produces distortion of fluid and electrolyte balance. Due to the loss of concentrating ability of the renal tubules as the load of substances to be excreted from the plasma has become greater,

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From the Section of Pediatrics, Mayo Clinic.
Read at the meeting of the Southern Minnesota Medical Association, Mankato, Minnesota, September 14, 1953.

removal of the load demands removal of larger volumes of water.

One sees then, that in advanced renal disease large volumes of urine of low specific gravity, and consequences of inadequate available water for renal function. In figure 3 the relationship of urinary volume to load of solute is depicted. On the right side of the graph the volume of water

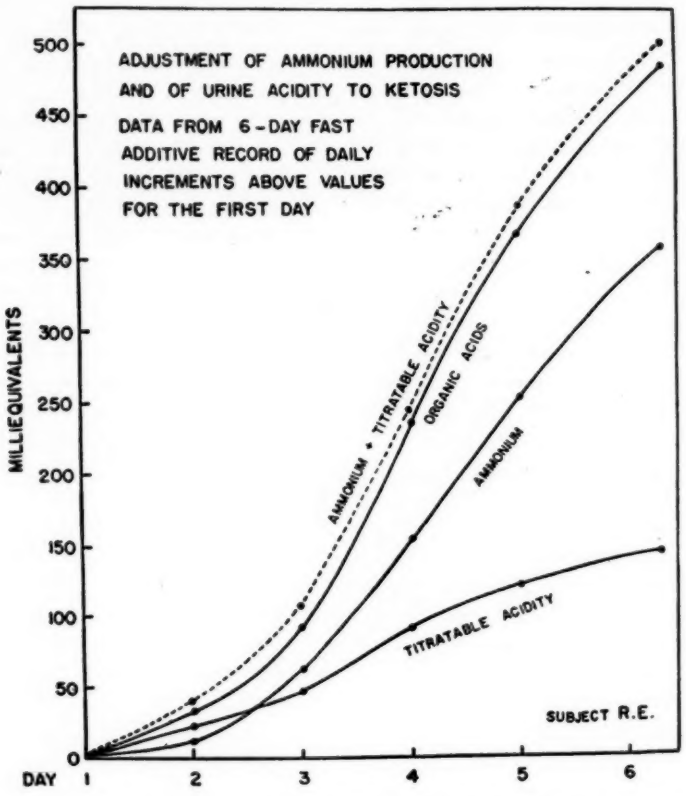


Fig. 1. Reprinted from "Companionship of Water and Electrolytes in the Organization of Body Fluids" by J. L. Gamble[5] with permission of the author and of the publishers, Stanford University Press. Copyright 1951 by the Board of Trustees of the Leland Stanford Junior University.

of alkaline reaction, are excreted.

Alkalosis is an unusual occurrence in renal disease, acute or chronic; therefore it merits no special consideration at this time.

The minimum amount of water required to form urine depends on the amount of substance or solute to be excreted. This need for water is usually defined as the renal obligatory water requirement. In children the renal obligatory need varies from 200 to 500 cc. per day. This requirement must be met even in starvation or thirsting and if not provided for by intake the need will be met by metabolism of the body stores of protein. Oliguria, anuria, or dehydration and death are the for intake and output is indicated for various routes.

To the daily water requirement for renal function must be added the volume of water lost through the lungs and skin. This is designated as the insensible loss of water. The 24-hour water requirement for children varies from 150 cc. per kilogram for the infant to 75 cc. per kilogram for the older child. Fever and high environmental temperature increase the daily water requirements. The importance of the consideration of water requirements and renal function will become evident in discussion of acute and chronic states of kidney disease.

### Acute Nephritis

The most common form of renal disease in children is acute hemorrhagic nephritis or glomerulonephritis. Varying degrees of severity of the

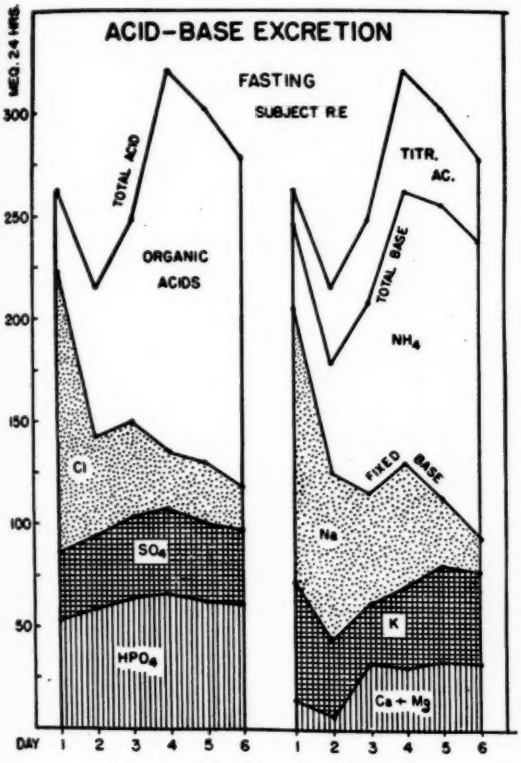


Fig. 2. (For legend, see Fig. 1.)

disease are known to occur but in general the recovery rate approaches 98 per cent.[6] Two-thirds of the cases of acute nephritis in childhood occur under the age of 7 years. The disease is rare in infancy. Most clinicians agree that there is a distinct difference in the prognosis for acute nephritis as it occurs in adults. Similarly, the nephrotic syndrome is a significantly different disease in the adult than it is in children.

Generally, the onset of acute nephritis follows a streptococcus infection and is heralded by hematuria. In the average case of acute glomerulonephritis there is no need for limitation of the intake of fluid, salt or protein; therefore the concern here will be with only two complications of acute nephritis associated with severe manifestations which demand special therapeutic considerations.

The first of the two types is referred to as "hypertensive encephalopathy." In this rather dramatic and acute illness the child suddenly has a convulsion. The blood pressure is markedly elevated and the child is comatose or confused. The concentration of blood urea usually is not elevated.

Prompt treatment to prevent convulsions and to reduce the hypertension is essential. Intramuscular administration of a 50 per cent solution of magnesium sulfate is effective as an anticonvulsant. The dosage is usually 0.2 cc. per kilogram of body weight, given every four hours until the adequate response in blood pressure is noted.

Magnesium relaxes arteriolar spasm to lower the blood pressure. Ettledorf and Tuttle have shown that it also increases the glomerular filtration rate, which is reduced in the acute phase of nephritis. The toxic effects of magnesium manifested by respiratory depression and narcosis can be counteracted by intravenous administration of calcium gluconate.

There is little need for rigid restriction of oral intake of fluids but the children frequently are comatose, in which case they require parenteral administration of fluid. Intravenous injection of fluid should be limited to an amount equivalent to the urinary output plus the estimated insensible loss. Sodium chloride should not be given, either intravenously or orally. The hypertensive phase usually abates within seven days and as a rule recovery is complete.

In the second group of patients the blood pressure may be found to be normal but oliguria and azotemia are apparent. The patients represent a small percentage of the total cases of acute nephritis but they are acutely ill and treatment is a special consideration. Oral intake of fluids may be limited somewhat and ingestion of salt should be restricted. As was mentioned previously, when oliguria or anuria is encountered intravenous administration of solutions must be restricted to replacement of daily losses from the skin, lungs and kidneys.

It used to be common practice to "flood" the anuric patient with parenterally given fluids to induce formation of urine and alleviate azotemia. The fallacy of this is obvious. Administration of an excess of fluid can only result in edema, dilution of the plasma electrolytes and aggravation of a serious clinical syndrome. Occasionally rapid administration of fluid results in convulsions. This occurrence has been termed "water intoxication."

Presumably, cerebral edema is the cause of the convulsions.

The value for serum potassium may rise sharply in anuric states to produce signs of potassium toxicity of hyperkalemia. The concentration of serum potassium is typically more than 5 mEq. per liter in this state and an electrocardiogram shows characteristic shortening of the Q-T interval, with marked peaking of the T wave. If sodium chloride in water is administered cautiously the effects of hyperkalemia or hyperpotassemia may be prevented.

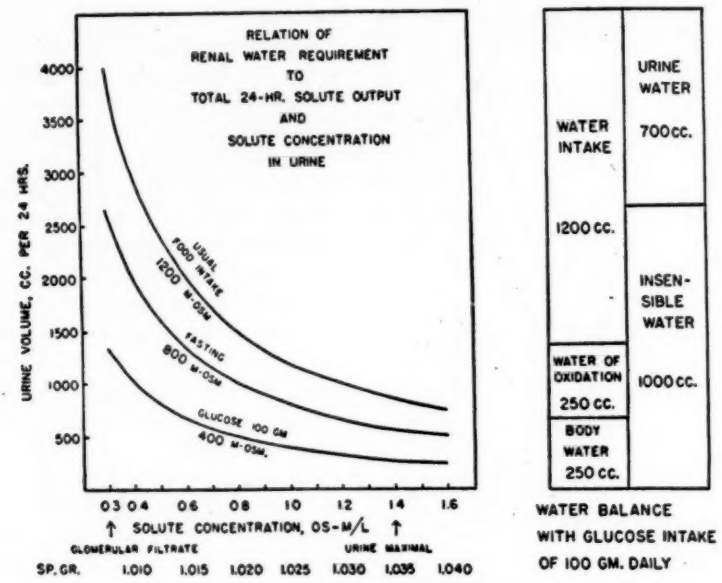


Fig. 3. (For legend, see Fig. 1.)

## Chronic Nephritis

Contrasted with acute nephritis, edema in chronic nephritis is exceedingly rare. The chronic phase of nephritis is unusual in children and there are differences of opinion as to the relationship of the acute nephritis of childhood to chronic nephritis in adults. Most likely there are rare cases of chronic nephritis of adults attributable to acute nephritis which occurred in childhood.

Edema[9] in chronic nephritis usually is found only as an accompaniment of cardiac failure. Proper treatment with digitalis is indicated, but restriction of the intake of salt must be cautious.

Characteristically, the patient with chronic nephritis excretes large volumes of dilute urine which has a low specific gravity and an alkaline reaction. The large output of urine is the result of diseased renal tubules. The glomeruli also are damaged; hence, azotemia is produced.

Tubular deficiency produces excessive loss of stores of fixed base; that is, sodium, potassium, calcium and magnesium. The tubule cell is unable to substitute hydrogen ions for base ions and an alkaline urine results. The production of ammonium by the tubule cells is deficient; therefore, large amounts of base are excreted with the acid radicals (sulfate, phosphate and organic acids).

The titratable acidity is absent in an alkaline urine with a pH above 7.4. The tubules also have been impaired to the degree that reabsorption of water is altered and the result is polyuria.

Gamble maintains that a high level of blood urea is protective to a certain extent in that this extra load of solute demands an added output of water for its removal. Oliguria or anuria following trial with the artificial kidney for removal of urea and azotemic products from the plasma has been commonly observed.

Loss of base in the urine over a prolonged period results in the mobilization of calcium from bone with consequent demineralization of the skeleton. Renal rickets, though not a common

accompaniment of this type of tubular disease, can occur and tetany may be noted.[2]

Albuminuria contributes to the possibility of tetany since calcium is in part protein-bound. Excessive treatment with alkali to correct the acidosis of renal disease may also produce tetany because of the reduction of available ionized calcium in an alkaline medium.

The treatment of chronic nephritis therefore consists in the provision of an adequate intake of water. Alkalinizing treatment with Shohl's solution (citric acid, 190 cc.; sodium citrate, 90 cc.; water sufficient to make 1,000 cc.), is indicated. The usual oral dosage of this mixture is 1 ounce four times a day.

Calcium may be supplied by a large intake of milk. Very little calcium will be absorbed from the intestine if any calcium salt other than calcium lactate is used. To be effective, calcium lactate must be dissolved in boiling water and cooled.

Some clinicians advocate the use of antacids on the theory that antacid blocks the absorption of phosphates from the gastrointestinal tract. Caution must be used in prolonged use of antacids, and occasional determinations of the pH or carbon dioxide content of the serum should be made to avoid gross disturbances of electrolytes.

While death from renal insufficiency is the usual outcome of chronic nephritis, many years may be added to the patient's life by careful management of the disease.

### Nephrotic Syndrome

The term "lipoid nephrosis" has been supplanted by the term "nephrotic syndrome." The latter is preferable because the clinical course of each case is similar to that of every other. Pure or lipoid nephrosis is less commonly encountered and supposedly is not accompanied by hematuria or azotemia. The diagnosis "nephrotic syndrome" applies to cases with or without microscopic hematuria, casts, albuminuria and mild azotemia.

Typically, the onset of the nephrotic syndrome is insidious, with edema of the periorbital region or of the ankle as the presenting symptom. From the onset the syndrome progresses to massive edema, ascites, hypoproteinemia, oliguria and anemia. Remissions and exacerbations are the rule and the predisposition to intercurrent infection is well known.

Kohn and Obrinsky's series of nephrotic patients has been followed over a period of twenty-six years. This series included fifty-seven children of whom thirty-six died. Most of the fatalities occurred before the advent of satisfactory treatment with sulfonamides and antibiotics.

In a more recent survey of cases by Philips, Calvin and Kagan, twenty-nine patients were followed from 1944 to 1950 and a more encouraging report is presented. These authors advanced the idea that the survival of twenty-three of the twenty-nine patients was primarily due to the prevention of infection. Four of the six children who died succumbed to bronchopneumonia. The importance of effective use of antibiotics and chemotherapy is apparent.

The physiologic alterations in the nephrotic syndrome are loss of protein through the damaged glomerular basement membrane, a decreased glomerular filtration rate and excessive retention of salt and water by the renal tubules.

In the past few years, significant developments in the treatment of the nephrotic syndrome have resulted through the use of corticotropin (ACTH) and cortisone. While foremost authorities in study of the nephrotic syndrome make no claim that these potent hormones cure the disease, there is increasing evidence that corticotropin and cortisone are effective in prolonging remissions. From this clinical observation has come careful speculation that the ultimate outcome will be a higher incidence of complete recovery.

In the past several years, the general attitude of pediatricians has changed from one of "let nature take its course" to one of more active treatment of the nephrotic syndrome. Dextran, a polysaccharide of high molecular weight and polyvinylpyrolidon (PVP) also have been employed as effective means of producing diuresis.

Metcoff and associates have advocated a ten-day course of corticotropin in a dosage of 150 mg. per square meter of body surface per day to be given in four divided injections to effect diuresis. Approximately 70 per cent of children so treated will undergo diuresis following this program. On a second, later trial diuresis will result in an additional 13 per cent. Other investigators have advocated oral administration of cortisone for several days a week for a period of six to eight weeks following diuresis.

The electrolyte pattern of the nephrotic syndrome has been well defined by the studies of Metcoff and associates. Before diuresis with cor-

ticotropin, the concentration of sodium, potassium, chlorine and carbon dioxide in the serum were normal. Following diuresis, no change of significance occurred except for a minor rise in serum sodium and a slight fall in serum potassium. Extensive studies of renal function showed that the diuresis accompanied significant improvement in the glomerular filtration rate and in renal tubular function. A few patients experienced gross disturbance of electrolytes which demanded prompt treatment of alkalosis. Several other patients suffered infections. The intake of fluid or salt was not restricted during treatment. Excessive salt was not permitted in the diet, however.

In the cases of Metcoff and associates, when sodium was needed potassium also was administered despite the lack of excessive loss of potassium during diuresis.

Those with whom I am associated believe that definitive treatment for patients with the nephrotic syndrome requires a course of corticotropin early in the disease, followed by oral administration of cortisone three days per week, in a dose of 100 to 200 mg. per day for a period of six to eight weeks after diuresis. It is essential that prophylaxis against infection with antibiotics be offered during treatment with corticotropin. Penicillin should be given orally during treatment with cortisone.

There is no need for rigid limitation of intake of fluid in remissions and a general diet without "added salt" may be employed. Barnett expressed the belief that there should be as few hospitalizations in the course of the disease as possible, and that activity should be restricted as little as practicable. As regards the use of antibiotics, Barnett has advocated liberal use of these agents when threats of infection occur but he has cautioned against continual administration.

Brief mention should be made of two additional syndromes wherein renal failure is concerned, and their associated fluid and electrolyte problems. The first of these syndromes is lower nephron nephrosis. This condition may be the consequence of various insults to renal integrity such as transfusion with incompatible blood, infection and shock. Considerable damage to the renal tubule cells occurs and tubular and epithelial casts appear in the urine. Anuria follows during the period of regeneration of the tubule cells.

Administration of fluids must be held to the replacement of the daily estimated losses until renal function is resumed. A period of four to seven days usually is required for recovery.

A second condition is the low-salt syndrome which occurs as a result of an inadequate intake of sodium. Edema is apparent and renal function ceases. Prompt replacement therapy with hypertonic sodium chloride, in 2 per cent or 6 per cent solution, is effective in restoring renal function.

## Summary

Only in anuric or oliguric states associated with renal disease should intravenous administration of fluids and salt be rigidly restricted. Fluids, when ingested orally, seldom produce aggravation of oliguric or anuric states. In chronic nephritis, if edema is absent, the intake of salt and water need not be restricted. Slight hope is offered for increased recovery rates among patients who present the nephrotic syndrome, through judicious use of corticotropin and cortisone. Additional help toward improvement of recovery rates can be expected from the antibiotics. Complete exclusion of salt from the diet alone can produce edema and oliguria. Treatment of the low-salt syndrome requires administration of hypertonic salt solutions.

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# CHEMOTHERAPY OF THE LEUKEMIAS AND LYMPHOMAS

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A FEW YEARS AGO a title such as this would have been meaningless because the treatment of leukemias and lymphomas was confined to roentgen therapy. Such treatment is still the standard by which any other must be measured, but at present several drugs have earned a place in everyday treatment and many others are under investigation.

## Mustards and Related Compounds

Since World War I, the mustard gases have been known to depress lymphocytic tissue. As the result of investigation during World War II, the nitrogen mustards were selected for clinical trial in the treatment of neoplastic diseases. Methyl-*bis* ($\beta$-chloroethyl)-amine, referred to as $HN_2$, has proved least toxic and easiest to manage. It is clinically effective in Hodgkin's disease, lymphosarcoma and leukemia. $HN_2$ probably exerts its action by interfering with mitosis and is similar in action to roentgen therapy. $HN_2$ is soluble in water but disintegrates rapidly; therefore, it must be given immediately after being dissolved. Because $HN_2$ may produce thrombophlebitis with ease, it is wise to inject this material into the tube of an intravenous infusion set in which a 5 per cent solution of dextrose is being used. The dose per course consists of 0.3 to 0.5 mg. per kilogram of body weight, the average total dose being 25 to 40 mg.; it is given usually in two to four divided doses. The immediate reactions are nausea, vomiting, fever and headache. Sedation with barbiturates before and after the injection and the use of dimenhydrinate (dramamine) may help to modify these reactions. Depression of the neutrophils and platelets may occur in one to twenty-one days. Seldom is this severe enough to be troublesome, although blood transfusions and antibiotic therapy occasionally are necessary. A course of $HN_2$ may be repeated in four to six weeks.

This drug has been most effective in the treatment of Hodgkin's disease. Remissions such as occur after roentgen therapy are common, but on the average they are not maintained for so long a period. The best indication for treatment with $HN_2$ is widespread and systemic involvement. The more localized lesions of Hodgkin's disease are better treated with roentgen therapy unless viscera are involved. $HN_2$ has been effective when radiation has lost its effectiveness; at times, tissues that have become resistant to radiation appear to respond again after treatment with $HN_2$. The value of combined treatment has not yet been determined. Evidence exists to indicate that the results of treatment with $HN_2$ followed immediately by roentgen therapy, may be superior to the results of either agent used alone.

The mustards are somewhat less effective in the treatment of other lymphomas but what has been said about Hodgkin's disease applies to them. Although improvement may follow the treatment of chronic leukemia with $HN_2$, the drug has been used only occasionally in this disease. It is of no value in the treatment of acute leukemia.

A closely related compound is triethylene melamine (TEM—Lederle Laboratories Division, American Cyanamid Company). Its effect on neoplastic tissue appears to be identical to that of $HN_2$ but it can be given orally and is effective in smaller doses. The standard dose is 5 or 10 mg.; it is given in the morning before the patient has had anything to eat or drink. Nausea and vomiting may occur but are not common. Administration is not repeated until the effect of the preceding dose is ascertained. Daily determinations of leukocytes and platelets are essential because some patients, particularly those who have lymphatic leukemia and lymphosarcoma, are extremely sensitive to this drug. Patients who have Hodgkin's disease or chronic myelogenous leukemia tolerate larger doses. A maintenance dose of 5 mg. every seven to ten days is usually possible but careful adjustment of the dose is required in each patient. Severe depression of the bone marrow may occur rapidly and renal damage also may result.

Another group of compounds, the triethylene-phosphoramides (TEPA—Lederle Laboratories Division, American Cyanamid Company) have similar properties and belong in the mustard

From the Section of Medicine, Mayo Clinic.

group. One of these, triethylene thiophosphoramide (thio TEPA), is fairly stable in solution and may be given orally in doses of 10 mg. It appears to have a wider margin of safety than does TEM. Its effectiveness has not been fully evaluated. It is of no value in the treatment of malignant melanoma.

Drugs of the mustard group have been used in the treatment of polycythemia vera, mycosis fungoides and metastatic carcinoma of the breast with variable but sometimes good results.

### Antimetabolites

Another group of drugs that act in a different way has found a place in the management of leukemia. These agents are the antimetabolites, the best known of which are the folic-acid antagonists. Of many such compounds, 4-aminopteroyl glutamic acid (aminopterin) and amethopterin are now in general use and are available. Unlike most of the other drugs discussed in this paper, these agents are of value in the treatment of acute leukemia. These drugs are extremely toxic and easily produce signs that resemble acute deficiency of folic acid. Administration of citrovorum factor or folinic acid will counteract this reaction. Most workers agree that toxic effects and remissions in acute leukemia are closely related, so that these drugs must be given until the toxic state is at least closely approached. The type of leukemia that responds most often is acute or subacute lymphocytic leukopenic leukemia of childhood or adolescence. All types of acute leukemia, however, have responded and occasionally an adult will be helped.

Aminopterin is given in doses of 0.5 to 1.0 mg. and amethopterin in doses of 2.5 to 5.0 mg. daily. It is important to watch for signs of toxicity during administration of folic-acid antagonists. Alopecia is annoying but not serious. Stomatitis with shallow ulceration is often the first sign of toxicity and soon may be followed by severe abdominal pain accompanied by bloody diarrhea. The latter manifestations are caused by ulcerative enteritis, which is usually of grave prognostic significance. Severe depression of the bone marrow with neutropenia, leukopenia and thrombocytopenia may occur but often are also part of the natural course of the disease. Stomatitis or abdominal pain is an indication to discontinue administration of the drug or reduce the dose. As previously mentioned, folinic acid in doses of 1 to 3 mg. may be used to counteract the toxic effects. When any improvement is to occur, reticulocytosis and regeneration of erythrocytes in the bone marrow often precede the disappearance of leukemic cells. About 25 per cent of children who have acute leukemia undergo remissions after use of these folic-acid antagonists; the average remission lasts for three months. However aminopterin and amethopterin are difficult to handle, and in many instances an exceedingly sick patient is made even more uncomfortable.

Folic-acid antagonists may be used in the treatment of chronic leukemia and the lymphomas but the hazards do not justify such use because other methods are available.

Another drug that probably should be classified as an antimetabolite is also useful in the treatment of acute leukemia. It is 6-mercaptopurine (purinthol—Burroughs Wellcome & Co.), which apparently is an antagonist of hypoxanthine and adenine. It is given in a dose of 1 mg. per pound of body weight and is administered orally in tablets containing 50 mg. each. Although nausea and vomiting may occur after its use, stomatitis is rare. The incidence and duration of remissions produced by this drug are not yet fully known.

### Hormones

Cortisone and corticotropin also have found a place in the treatment of acute leukemia. The doses used are large; 200 mg. of cortisone or 100 mg. of corticotropin is given as long as these agents are tolerated. Lymphocytic leukemias are most likely to respond and again most of the remissions occur in children. While use of these steroids induces good remissions in about 25 per cent of patients, the duration of the remissions averages about half of that obtained with the folic-acid antagonists, namely, six weeks instead of three months. One advantage of these hormones is their absence of severe toxic reactions. The folic-acid antagonists may be given simultaneously with the hormones or alternate courses of each agent may be used.

Cortisone also may be used in the treatment of Hodgkin's disease, in which it causes shrinkage of involved tissue but only short remissions. In chronic lymphocytic leukemia it may favorably affect the cutaneous lesions even though the numbers of blood cells are not altered. It also should be tried in hemolytic anemias secondary to lymphoma or chronic leukemia.

### Miscellaneous Drugs

Urethan (ethyl carbamate) has been used in the management of chronic leukemia, particularly myelogenous leukemia. Administration of doses up to 3 gm. per day produces a decrease in the number of leukocytes and in the size of the spleen. The great difficulty with this drug is the nausea and vomiting which it may induce; however, restriction of doses to 1 or 2 gm. per day usually prevents this effect. My experience, however, has been that urethan is not so effective in inducing and maintaining remissions in chronic myelogenous leukemia as is roentgen therapy; it offers little benefit when the disease is radioresistant.

English workers recently have introduced a new compound, namely, 1,4-dimethanesulfonoxybutane (myleran—Burroughs Wellcome & Co.), which is reported to be effective in the treatment of chronic myelogenous leukemia. While my experience indicates that the drug may reduce the number of leukocytes, I cannot say whether it will prove more useful than urethan.

Many years ago, Fowler's solution was used in the treatment of leukemia, particularly in the chronic myelogenous type. Although this preparation is subject to the disadvantages of arsenical therapy, it is still a useful drug in this disease.

### Comment

My colleagues and I have noted, as have others, that multiple remissions may occur in acute leukemia when the same drug is used on different occasions and that leukemia which becomes resistant to one drug may respond later to another. While such occasions are not common, they do occur. At all times, general supportive care, such as blood transfusion and antibiotic therapy, is carried out. While these factors may influence survival times, I do not think they are responsible for remissions. It must be emphasized that no curative agent for acute leukemia has been found and that remissions in children are not the rule and are rare in adults. At the present time I consider that the best program of treatment, if the patient or the parents understand the whole picture and still desire to have such treatment, is a trial of a folic-acid antagonist combined with or followed by use of corticotropin or cortisone. Administration of 6-mercaptopurine may be of value when the disease is resistant to use of folic-acid antagonists. It is questionable whether any drugs are useful in patients more than thirty years of age.

The availability of drugs that will augment or supplement roentgen therapy in Hodgkin's disease, the lymphomas and the chronic leukemias should be welcomed by the general practitioner, the pediatrician and the internist. It means that renewed interest may be taken in patients, formerly dismissed to the care of the radiologist and it should lead to closer co-operation between the family physician and the specialist. This should result in better care for the patient.

### Summary

Roentgen therapy remains the standard treatment for Hodgkin's disease, lymphoma and chronic leukemia.

Nitrogen mustard is effective in the treatment of these diseases, particularly Hodgkin's disease. Related compounds that may be given orally are triethylene melamine and triethylene thiophosphoramide. In selected cases they are useful agents.

The folic-acid antagonists, as well as corticotropin, cortisone and 6-mercaptopurine cause remissions in acute leukemia in children, and young adults but such remissions are not long maintained.

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### SCHOLARSHIPS IN PUBLIC HEALTH

The Harvard School of Public Health is initiating postgraduate scholarships in amounts ranging up to $5,000 to qualified candidates desiring to study at the school during the academic year 1954-1955. These scholarships are available to (1) physicians, dentists, and veterinarians interested in preventive medicine and seeking training in one or more public health specialties leading to a master of public health or a doctor of public health degree; (2) industrial physicians seeking training in industrial medicine leading to a master of industrial health degree; (3) public health nurses with a college degree and satisfactory field experience who wish additional public health training leading to a master of public health degree; (4) public health engineers seeking additional training and research experience in one or more public health specialties leading to either a master of science in hygiene or a doctor of science in hygiene degree; and (5) college graduates with academic experience in the natural sciences who desire training and research experience in one of the sciences related to public health and leading to either a master of science in hygiene or a doctor of science in hygiene degree. Applications must be filed by April 30. Winners will be announced June 1. Information may be obtained from the Secretary, Harvard School of Public Health, 55 Shattuck St., Boston 15.

# Laboratory Aids to Medical Practice

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## OFFICE HEMATOLOGY

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**La Crosse, Wisconsin**

"OFFICE HEMATOLOGY" refers here to routine blood tests performed on outpatients *in the doctor's office.* Few physicians can obtain or maintain a registered medical technologist. Many rely on the "office nurse," who may be neither nurse nor technician. The doctor himself, therefore, is responsible for the reliability of his office laboratory. Unreliable laboratory data are worse than none at all!

Office hematology should provide adequate screening for primary hematologic disease and should furnish dependable quantitative information for clinical evaluation and treatment of secondary hematologic problems. Realize the limitations of the office laboratory and freely seek hematologic consultation. Do not be guilty of the diagnosis of anemia by therapeutic trial. Have respect for persistent leukopenia with anemia. Use caution in diagnosis and prognosis of adenopathy with lymphocytosis in children and young adults; in such cases, send blood for determination of heterophil antibodies once a week for three weeks and send good blood smears to a clinical laboratory.

Choose office blood tests for maximal dependable information, rapidity and minimal cost but never trade time for accuracy. A single determination has much less significance than a series, which may show a "trend." Repeat tests whose results are inconsistent with clinical impressions. No one hematologic finding is pathognomonic. A minimal hematologic screening for every new office patient should consist of: (1) hemoglobin or hematocrit; (2) total leukocyte count and (3) peripheral blood smear. An erythrocyte count gives little information and has great personal and technical error; it may well be omitted unless cell indices are to be done. The erythrocyte count may be normal in hypochromic anemia. The hematocrit is more valuable than either hemoglobin or erythrocytes in following the treatment of anemia.

I would do a differential count only when indicated by the number of leukocytes or the appearance of the blood smear. The differential count requires a well-stained smear and proper training in morphologic hematology or it is worthless. I often receive blood smears for diagnosis accompanied by total and differential leukocyte counts, yet find the smear so poor that I cannot identify cells for a differential count, to say nothing of returning a morphologic diagnosis.

Depending on the size of the office laboratory and on trained personnel, one could set up certain unit or group tests to be done on the basis of clinical diagnosis and results of basic screening tests. For the study of anemias, such a unit might include: (1) complete blood counts; (2) hematocrit; (3) cell indices and (4) reticulocyte count. For a study of hemorrhagic tendencies the unit also would include: (1) bleeding time; (2) clotting time; (3) clot retraction; (4) platelet count and (5) prothrombin time.

### Hemoglobinometry

Determination of hemoglobin is the most commonly used of all office blood tests, but it also has the greatest range of error (5 to 40 per cent). Error is referable to: (1) differences in methods and large inherent error; (2) lack of uniform standard of reference; (3) pipette and instrument error; (4) personal technical errors in visual colorimetric methods and (5) instability of color of hemoglobin and acid hematin.

The Tallqvist scale and Dare method have the

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This is the fourteenth in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

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greatest inherent error. The popular Sahli method with careful technique and calibrated pipettes and tubes can attain a satisfactory accuracy of ±5 per cent. The following precautions reduce technical error in the Sahli method: (1) use a *dry,* bubble-free pipette; (2) use clean diluting fluid; (3) wipe off blood on outside of pipette; (4) fill pipette accurately (if overdilution occurs, discard and start over); (5) discard first drop of diluting fluid; (6) rinse acid in Sahli tube back and forth several times; (7) stir constantly while delivering pipette sample into Sahli tube; (8) allow a minimum of twenty minutes at room temperature before diluting the acid hematin to match color standard; (9) stir while diluting and match color in same good light each time; (10) read meniscus in Sahli tube in grams per 100 cc. of blood and (11) use tubes made by same manufacturer as standard.

The Haden-Hausser hemoglobinometer (larger laboratory model) has the advantage of allowing use of the blood sample in the white pipette. It also allows approach of the end point from either side without the danger of passing it. Both Sahli and Haden-Hausser techniques are visual colorimetric acid-hematin methods subject to inherent errors of fading and the use of artificial permanent color standards. Each physician must determine, for the instrument used, the equivalent in grams per 100 cc. for 100 per cent hemoglobin on a series of healthy, normal adults with erythrocyte counts near 5,000,000. A value of 15 gm. per 100 cc. of blood is the average acceptable standard for this region.

### Hematocrit

The volume per cent of packed erythrocytes is the most accurate index of circulating hemoglobin and erythrocytes. The hematocrit is quantitative, simple, informative and does not require unusual technical skill. A good method is that of Wintrobe, as detailed in Wintrobe's *Clinical Hematology.* The equipment required includes Wintrobe hematocrit tubes, capillary pipettes and any standard centrifuge capable of 2,500 to 3,500 rpm. Some precautions for accurate results include: (1) dry anticoagulant in clean tubes; (2) invert blood sample in oxalate tube ten to twelve times; (3) fill hematocrit tube immediately after mixing; (4) let blood settle in hematocrit tube for one hour before centrifuging (this gives sharper demarcation of layers and also permits a sedimentation rate reading in the tube); (5) centrifuge for thirty minutes and (6) use hand lens to read level of packed erythrocytes.

The normal hematocrit value in men is 47 ± 7 per cent and in women is 42 ± 5 per cent. In anemia, the value may go as low as 8 per cent, whereas in polycythemia it may be as high as 80 per cent.

### Blood Cell Counts

A likely source of error in cell counts is wet pipettes. A dry pipette is one in which the bead is free in the bulb. The best way to assure a dry pipette for each count is to have enough on hand for the entire day's use without employing any pipette twice. A clean dry counting chamber is also imperative. Discard pipettes with even a tiny chip at the tip. We have greatly reduced technical errors in dilution by replacing all conventional red and white pipettes with Tri-Lyne pipettes. Bard-Parker pipettes probably are the best of the conventional types because they are calibrated individually and marked with a correction factor. The counting chamber must not be overfilled or underfilled for an accurate count.

### Blood Smears

These are the most important and also the poorest done of all office hematology procedures. Any smear might be the one you want to send to a pathologist or hematologist. With a little personal supervision, Wright-stained blood smears done in the office can be just as good as those done in the clinical laboratory.

Clean slides are essential. Slides in a freshly opened box are not clean. Wash up a batch in ordinary soap and water. Rinse in plenty of hot clean water, with a final rinse in distilled water. Store in 95 per cent alcohol (ethyl) until ready for use. The best smears use a small drop of blood (pinhead size) spread lightly, evenly and without a final push or lift. Dry smear rapidly in air; do not blow or use a fan. High humidity in summer makes poor smears and poor stain affinity. I know of no remedy. Use only certified Wright stain (prepared or powder). If you make your own, use only acetone-free methyl alcohol. Keep stock Wright stain in tightly stoppered bottle. Dispense it with a small dropper bottle. Filter stain each day. Keep bottle of stain away from strong acids or alkalies.

*(Continued on Page 292)*

# President's Letter

## WE MEET AGAIN

The tradition of fine medical meetings will be carried on again in 1954. The annual meeting of the Minnesota State Medical Association is scheduled for Duluth on June 7, 8, and 9, and from all indications, the convention promises to be outstanding in all respects.

The scientific program will be one of unusual value to the medical profession of this area, specialist and general practitioner alike. Subjects run the gamut of medical interests, including such worthy subjects as ulcerative colitis, a symposium on fractures, a symposium on cortisone and ACTH, treatment and diagnosis of hypertension, diagnosis of pulmonary diseases, a symposium on anesthesiology, hydronephrosis, treatment of blood dyscrasias, anemias in pregnancy, plasma expanders, and a section on what's new in medicine, surgery, radiology, pediatrics, obstetrics and gynecology.

The usual wide selections in subject matter will again be found in the round-table discussion luncheons. Many guest speakers from the general session will lead these discussions, giving expert advice and opinion to the physicians who look on the annual meeting as an excellent opportunity to gain additional information as an adjunct to their everyday practices.

Assurance of excellent hotel accommodations has been given by the Duluth Chamber of Commerce.

Business sessions scheduled during the meeting include the following. The Council meets daily, beginning on Saturday, June 5. The House of Delegates will begin its sessions on Sunday, June 6. Association committees will meet on Tuesday and Wednesday, June 8 and 9.

A new feature this year will be one or two "consultation" booths, to be found on the convention floor at the Duluth Armory. Plans include one booth on general fracture subjects, another on obstetrics and gynecology. Here, the physician can bring his own special problems, outline them to experts in the field, and discuss their implications and treatment.

Many important social events have been planned. Special organizations will be meeting simultaneously with the State Association, holding special luncheons and dinners for their members.

Recreation has been arranged by the Committee on Local Arrangements. Golf, lake fishing and skeet and trap shooting will give the busy doctor a fine chance for relaxation away from the heavy schedule of scientific and business sessions. Duluth offers many good accommodations for such affairs, and physicians are urged to take time out to enjoy them.

There are many things one might mention in giving a general outline of the meeting of the year. With all the various events scheduled, it is a matter of choice on what to concentrate. But there is one important thing to remember: your attendance at the meeting in Duluth will be well worth the few days away from your practice. It offers postgraduate education from experts in all fields; it offers meetings with your special interest group; it offers time for relaxation with your colleagues throughout the profession; and it offers opportunity to meet and discuss Association problems and policy which are common to the nation's entire medical profession.

For interesting and informative medical sessions, your meeting calendar should be marked for the Minnesota State Medical Association annual convention, Duluth, Minnesota, June 7, 8 and 9, 1954.

J. Chase.

*President, Minnesota State Medical Association*

# Editorial

ARTHUR H. WELLS, M.D., *Editor;* HENRY G. MOEHRING, M.D., and JOHN F. BRIGGS, M.D.

## CANCER DETECTION COST vs. VALUE OF LIFE

AFTER almost twenty years of experience with Cancer Detection Centers, the medical profession should soon be able to properly evaluate many of the practical aspects of these periodic examinations of apparently healthy individuals. Perhaps the closing of large numbers of the Centers throughout the country and the recent shift of emphasis by the American College of Surgeons to Cancer Diagnostic Clinics (patients come with complaints) are signs of practicality.

Three principal questions have confronted the profession. (1) Which of the individual tests performed in the examinations is worth its salt in terms of lives saved per dollar spent? (2) Is the specialty clinic approach to cancer detection more economical than the "Every Doctor's Office a Cancer Detection Center" plan? (3) What new tests for cancer detection are there to be discovered? The answer to the last question could make almost any amount of money expended in this type of research worthwhile.

There is some value in nearly all of the clinical and laboratory procedures generally performed in these examinations. The question is, are they economically sound? An x-ray examination of the chests of more than a million people in the Los Angeles area was worth the cost in numbers of cases of tuberculosis discovered, isolated and treated. On the other hand, if the study had been carried out solely for the purpose of discovering bronchogenic cancer, rough calculation leads one to the conclusion that it would cost approximately $100,000 per life saved from this disease. As a matter of fact, there is some question as to the economy of any type of roentgen study conducted solely for the detection of cancer in the symptomless and signless individual.

From the figures of 11,000 examinations for cancer presented by Hitchcock and Aust elsewhere in this issue, one can see that only ten possible curable, asymptomatic cancers were found which were not easily accessable to the examining fingers and eyes of a physician in general practice. The salvage from these ten cases of deep-seated cancer may have been five lives. Had the ten eventually developed symptoms and gone to their family physicians in the conventional manner, at least two would have been saved. Would the difference of three lives cost an additional $50,000? We are badly in need of a dollars and cents approach to this subject concurrent with the scientific study.

Physicians are on solid economic ground when the cancer detection (in the apparently healthy person over forty years) is limited to a study of cancer symptomatology and a careful physical examination including cervical and rectal visualization. Any clues of "internal" cancer picked up in the clinical history must receive the proper diagnostic attention. The corrected cost of this type of cancer detection will be between $1,000 and $2,000 per life saved from cancer. In addition, there will be a bountiful disclosure of "precancerous" and non-neoplastic diseases. The "Every Doctor's Office . . ." program of your Committee on Cancer deserves the whole-hearted support of the profession.

## BLUE SHIELD'S PLAN B

CONCLUDING that open, written debate on important issues serves the profession best, your Board of Editors publishes the following editorials on the Pro and Con of Plan B of Blue Shield for your careful consideration.

### PRO

Both Blue Shield and Blue Cross were organized to provide people with a means of paying their medical-care and hospital bills at a cost within their financial reach. During the depression of 1932, many people throughout the United States were unable to pay their hospital bills, as a result of which the occupancy of hospitals dropped to a hazardously low level. To remedy this situation, Blue Cross devised a plan whereby it accepted small monthly payments from those who joined the movement for the payment of their hospital bills. It was a non-profit, prepayment plan to provide hospital care at a cost which people could afford.

Similarly, the depression of 1937 created serious problems in the payment of medical care bills.

Many people were unable to pay their doctor bills, and both the medical profession and the government became interested in what was referred to as "the high cost of medical care." The government's solution appeared in the form of the first Wagner-Murray-Dingell bill introduced in Congress in 1939. The medical profession's answer appeared the same year in the organization of the first Blue Shield medical-care plan fashioned after the Blue Cross hospital plan.

In this problem of the so-called "high cost of medical care," classification of people according to their incomes and their ability to pay for medical care has been necessary. Indigent persons receive their medical care at the cost of some unit of government, local, state or federal, and, consequently, are not concerned in pre-payment plans. The wealthy are able to pay the costs of their medical care, and do not constitute part of the problem. This leaves those in the low and modest income brackets as the ones who must be provided medical care at a cost they can afford. If medicine, through Blue Shield or some other equally effective means, does not furnish the answer of providing medical care for those with low or modest incomes, the solution might be forthcoming from government, labor or industry. Thus, Plan B has the rôle of furnishing part of the answer by offering medical care to those with modest incomes within the reach of their pocketbooks.

As a first step in the development of Blue Shield's Plan B, the issuance of a contract with service benefits for families with annual incomes less than $5,000 was first discussed with the Council and the Houe of Delegates of the Minnesota State Medical Association at the Radisson Hotel in May, 1952. No action was asked nor taken regarding it, but neither were any adverse comments made regarding it. The matter was again presented to the Council at the meeting of the Minnesota State Medical Association in May of 1953. At this time, the contract's increase of fees to approximately 50 per cent more than those of the standard contract, and the contract's income limits for service benefits of $3,600 for single persons and $5,000 for families were fully presented. No objections were raised at the Council meeting, and the matter was referred to the House of Delegates. Here again, these same two features of Plan B were clearly discussed before the House of Delegates, which then approved the plan with only one dissenting vote. Plan B, then, was developed only after it was approved by the governing body of organized medicine in Minnesota.

After the announcement of its issuance, two objections to Plan B were voiced by some members of the medical profession. The first of these concerns fees. As a first step in the deterimination of an adequate fee schedule, a questionnaire was sent to the component medical societies asking them to list the fees customarily charged by their members for some of the more common medical-surgical-obstetrical services. Replies were received from nineteen of the thirty-two component medical societies. Then, four of the highest Blue Shield fee schedules of such higher income contracts as Plan B were assembled. The fees selected for the Plan B schedule consisted principally of the highest or next highest fees of the four Blue Shield schedules. By comparison with the fees listed in the questionnaires, the Plan B fees were usually above those customarily charged by members of the various component societies. The final Plan B fee schedule has fees, in general, 50 to 55 per cent higher than those of Plan A. Also, the Plan B fee schedule ranks among the highest Blue Shield fee schedules in the United States.

Despite its generally favorable comparison with other fee schedules, it has been pointed out that it still contains some inequities. To overcome these objections, the Board of Directors has appointed a special committee whose purpose it is to meet with the authorized representatives of any group of physicians for the review and adjustment of inequities. Efforts are now being made to adjust inequities of fees and other Plan B matters in this manner.

The second objection to Plan B is that Plan B might establish the fees for 75 per cent of the population. This statement is based upon the fact that an average of approximately 75 per cent of single persons and families in Minnesota have incomes less than the service income limits of the Plan B contract.

Enrollment under Plan B contracts in other states, as well as Plan A enrollment here in Minnesota, fails to support such a statement. In one of the states having such a contract, after several years of comparatively successful Plan B enrollment, only a relatively small percentage of the population had Plan B coverage. Were this same rate enrollment of Plan B to occur in

Minnesota, is it likely that Plan B might establish fees for 75 per cent of the population?

It has been calculated from income data available in 1949 that an average of approximately 70 per cent of single persons and families then had incomes less than the standard contract's service income limits. This is only five per cent less than the percentage now said to be potentially eligible for service benefits under Plan B, yet no statements have been made that the standard contract established the fees for 70 per cent of the population.

Other economic factors have an important bearing on these service income limits. For instance, since Blue Shield was organized there have been five general, national wage and salary increases. These salary raises have elevated the incomes of many workers to the point where they are no longer eligible for Plan A service benefits. One of the Building and Construction Trades Council in Minnesota lists twenty-nine types of construction workers whose lowest hourly wages make them ineligible for Plan A service benefits. The Agriculture Department and the Farm Management Office of the University Extension Service found the average annual farm labor income in the southern half of Minnesota in 1952 to be $3,950. Are not these wage earners and farmers the types and groups of people who were originally intended to have service benefits under the Blue Shield program?

Inflation also has developed to the point where the dollar today has only 60 per cent of its value in 1938. This means that a family with an annual income of $5,000 today has the same purchasing power as a family with a $3,000 annual income sixteen years ago. Families of three or four persons, and usually families in this income bracket are larger, are often unable to pay for medical and hospital care. Here again, it is apparent that these are the same people for whom Blue Shield's service benefits were originally planned.

Thus, Plan B is a necessary part of the answer to the problem. As a complement of Plan A, and with a catastrophic illness rider, Plan B provides the answer to the broad problem often referred to as the high cost of medical care. Plan A alone will not solve the problem. Plan B is an essential part of the program of providing medical care for people with low or modest incomes at costs they can afford. Recommendations have emanated from high authorities in both the past Democratic and the present Republican administrations that voluntary prepaid medical care plans provide greater benefits for more people. And, if this challenge is not met by the medical profession through such a medium as Minnesota Blue Shield's Plan B, there is little doubt that the answer may be forthcoming from some other source.

E. J. Simons, M.D.

**CON**

Blue Shield of Minnesota has rightfully claimed the support of the preponderant majority of physicians in Minnesota since its inception in 1947. Members of the State Medical Association have recommended this form of voluntary pre-paid medical insurance as the best buy in its field. The medical profession has supported the Blue Shield as its own medical insurance plan believing that it would materially relieve the financial burden of sickness upon Minnesota families.

The Blue Shield contract has proved its value in the succeeding years. It has earned the support of the public and the profession generally. Although in many instances the fees paid were inadequate, the physician dealing with a patient in reasonably good financial circumstances could ask for supplemental payment. Participating physicians were required to accept the Blue Shield payment as full settlement of medical charges for indigent and low income groups: single individuals with an income less than $2,000.00 per year or families with less than $3,000.00. Minnesota physicians participating in this plan subscribed to these limitations and restrictions. The profession accepted the inevitable inequities that occur when a third party sets the fee, occasionally providing medical care without fee in some cases which required the services of more than one physician. The percentage of cases falling into such a category was small.

This happy state of affairs has been severely disturbed by a letter sent out December 23, 1953, by Minnesota Medical Service Incorporated (Blue Shield), outlining a new plan, the Plan B contract, to be available to the public after March 1, 1954. Quoting from this letter:

"Service benefits under this Plan B contract will be available to single persons with annual incomes less than $3,600.00 and to family participant subscribers whose annual family income is less than $5,000.00 The Blue Shield allowances for eligible services provided the subscribers whose incomes are below the level stated for each type of subscribers—single or family—*will consti-*

*tute payment in full.* If the annual income is above these levels the subscriber is responsible for the balance of the doctor's usual fee."

This means that the Blue Shield allowance represents payment in full to the participating physician for the medical obligations of family subscribers with annual incomes not in excess of $5,000.00 per year and single subscribers having annual incomes not in excess of $3,600.00 per year. It is estimated that approximately 75 per cent of the gainfully employed people of the State of Minnesota have annual earnings within these income levels. It is reasonable to assume therefore that this plan, if it finds wide public acceptance, will in effect establish fees for 75 per cent of the population of this state.

Large segments of Minnesota physicians are alarmed by the introduction of this drastic change in Blue Shield coverage. The so-called full coverage under the proposed plan means absolute regulation of physicians' fees not only for its indigent and low income groups but also for the average man and necessarily for the above average as well! Inequities that were of little consequence affecting a small indigent group can no longer be ignored. In particular, the nonoperating specialties, internal medicine, dermatology, neuropsychiatry, pediatrics, and anesthesiology will suffer. However variously the new plan may create economic burdens, groups within the entire profession must suffer equally the loss of independence. Several groups have already voiced their opposition to the plan. Both the Minneapolis and Saint Paul Societies of Internal Medicine have had special meetings expressing their opposition. When the proposed plan was presented by officers of the Blue Shield recently before a special meeting of the Minneapolis Asbury Hospital Staff, every comment was critical.

It is evident that large sections of the membership of the Minnesota State Medical Association, probably the majority, are opposed to extension of the complete fee service to a $3,600.00 and $5,000.00 level.

It seems proper, therefore, to suggest to the Board of Directors that this is not an opportune time to inaugurate this new plan. It might be further suggested to them that they more carefully take soundings of medical opinion before making drastic changes in the Blue Shield contracts.

R. BERMAN, M.D.

## THE ANNUAL MSMA MEETING

The annual meeting of the Minnesota State Medical Association to be held June 7, 8, and 9 in Duluth shows every promise of being a successful one by everyone's standards. You can help make it successful by sending in your reservations for housing, for specialty meetings, and for special events *early.*

The hotel managers have given assurance that housing for the meeting will be even better than in 1950. You are urged to minimize any causes of discontent by:

1. Mailing or telephoning in your reservations early.
2. Stating your preference, and an alternate or two, of type of accommodations.
3. Stating your checking out time, if possible.

Since reservations will be held until 4:00 P.M., it is suggested that you send a deposit with your reservation request, if you think you will sign in *after* 4:00 P.M. (Deposit will be returned, if the reservation is cancelled before 4:00 P.M.)

# Minnesota State Medical Association

## 101st Annual Meeting

**Duluth, Minnesota**
**June 7, 8 and 9, 1954**

### *Preliminary Program*

## BUSINESS SESSIONS

### SATURDAY, JUNE 5
**Hotel Duluth**

2:00 P.M.—Council..... English Room
6:00 P.M.—Council.....English Room

### SUNDAY, JUNE 6
**Hotel Duluth**

8:30 A.M.—Council.....English Room
10:00 A.M.—Reference Committees.....
Rooms to be assigned
1:30 P.M.—House of Delegates.....Ballroom
8:00 P.M.—House of Delegates.....Ballroom

### MONDAY, JUNE 7
**Hotel Duluth**

8:30 A.M.—Council.....English Room
12:15 P.M.—House of Delegates.....Ballroom

### TUESDAY, JUNE 8
**Hotel Duluth**

8:30 A.M.—Council.....English Room
8:30 A.M.—Committees.....
Rooms to be assigned

### WEDNESDAY, JUNE 9
**Hotel Duluth**

8:30 A.M.—Council.....English Room
8:30 A.M.—Committees.....
Rooms to be assigned

## SOCIAL EVENTS

### SUNDAY, JUNE 6

**Buffet Supper**

**American Medical Women's Association, Inc., Minnesota Chapter**

6:30 p.m., Home of Elizabeth Bagley, M.D., 2524 Branch Street
Open to all visiting women doctors. Dr. Marie Bepko will show colored slides from the Fourth Congress of the Pan-American Medical Women's meeting in New York City.

### MONDAY, JUNE 7
**Luncheons**

**Minnesota Chapter, American College of Chest Physicians**

12:15 p.m.

**Minnesota Medical Foundation**

12:15 p.m.
Speaker: CLARENCE DENNIS, M.D., New York

**Dinners**

**Minnesota Academy of General Practice**

6:00 p.m., Ballroom, Hotel Spalding

**Open House, Minnesota State Medical Association**

9:00 p.m., Ballroom, Hotel Duluth

**Minnesota Radiological Society**

6:00 p.m., Social Hour
7:00 p.m., Dinner
Speaker: PHILIP J. HODES, M.D., Philadelphia—"An Evaluation of Body Section Radiography"
Northland Country Club, 39th Avenue East and Superior Street. Tickets, $5.00. Reservations should be made with Dr. John R. McNutt, 824 Medical Arts Building, Duluth.

### TUESDAY, JUNE 8
**Dinners**

**Minnesota Society of Clinical Pathologists**

6:00 p.m., Annual Dinner and Business Meeting—House of Dr. A. H. Wells

**Annual Banquet, Minnesota State Medical Association**

7:00 p.m., Ballroom, Hotel Duluth

## 12:15 ROUND TABLES

### Duluth and Spalding Hotels

### MONDAY, JUNE 7

Anesthesia as a Maternal Mortality Factor
FREDERICK J. HOFMEISTER, Milwaukee, Wisconsin

### TUESDAY, JUNE 8

Use of Tracheotomy in Chest Injuries
W. P. EDER, Minneapolis
Treatment of Burns in Children
TAGUE C. CHISHOLM, Minneapolis
Emergency Treatment of Nerve Injuries
ROBERT L. MERRICK, Saint Paul
Indications and Contraindications for the Use of the Hormones (ACTH and Cortisone) in Gastrointestinal Disorders
J. ARNOLD BARGEN, Rochester

Skin Manifestations in Systemic Disease
JOHN F. SCHMID, Duluth
Acute Urinary Obstruction in the Male
OTTO C. PHARES, Saint Cloud
Drugs in Peptic Ulcer
E. E. GAMBILL, Rochester
The Injured Hand
J. C. FEULING, Duluth

### WEDNESDAY, JUNE 9

Diagnostic Difficulties in Carcinomas of the Lungs
F. F. CALLAHAN, Saint Paul
Evaluation of Operations for Peptic Ulcer
C. W. LILLEHEI, University of Minnesota
Relation of Blood Volume to Gastrointestinal Hemorrhage
J. E. TWOMEY, Minneapolis
Practical Application of Cytologic Technique for Malignancy
A. H. WELLS, Duluth
Anesthesia in Tonsillectomy
F. C. JACOBSON, Duluth
Functional Uterine Bleeding
R. F. STURLEY, Saint Paul
Treatment of Plantar Warts
R. W. GOLTZ, Minneapolis
Diagnosis and Treatment of Blood Dyscrasias
PAUL S. HAGEN, Saint Paul

## GENERAL SESSION

**Duluth Armory**

**MONDAY, JUNE 7**

A.M.

8:30 Visit Scientific and Technical Exhibits.... Armory

9:00 TREATMENT OF HYPERTENSION
Moderator—R. M. SHICK, Rochester
HENRY A. SCHROEDER, Saint Louis, Missouri
A. C. CORCORAN, Cleveland, Ohio
ROBERT W. HOLLENHORST, Rochester
Question and Answer Period

**Intermission**

10:15 Visit Scientific and Technical Exhibits.... Armory

11:00 Plastic Operations for Hydronephrosis
FREDERIC E. B. FOLEY, Saint Paul
Comments
ORMOND S. CULP, Rochester

11:40 Staphylococcal Sepsis; Control with Antibiotics
ROBERT I. WISE, Assistant Professor, Department of Medicine, University of Minnesota

P.M.

12:15 Miscellaneous Luncheons
Round-Table Luncheon

1:30 Visit Scientific and Technical Exhibits..Armory

2:00 (To be announced)

2:30 RUSSELL D. CARMAN MEMORIAL LECTURE
Pitfalls in the Diagnosis of Pulmonary Diseases
PHILIP J. HODES, Professor of Radiology, School of Medicine, University of Pennsylvania, Philadelphia

**Intermission**

3:15 Visit Scientific and Technical Exhibits..Armory

4:00 SYMPOSIUM ON ANESTHESIOLOGY
Moderator—RALPH T. KNIGHT, Minneapolis
Hypotension During Surgery and the Immediate Postoperative Period
FERDINAND C. JACOBSON, Duluth
Cardiac Arrest During Anesthesia
JAMES H. MATTHEWS, Minneapolis
Will Analgesia Evolve from a Century of Anesthesia?
JOHN S. LUNDY, Rochester
Trichlorethylene—Its Possibilities for the General Practitioner
O. SIDNEY ORTH, Professor of Pharmacology, University of Wisconsin, Madison

5:00 Visit Scientific and Technical Exhibits..Armory

6:00 Special Dinners

9:00 Open House.....Hotel Duluth

## GENERAL SESSION

**Duluth Armory**

**TUESDAY, JUNE 8**

A.M.

8:30 Visit Scientific and Technical Exhibits..Armory

9:00 SYMPOSIUM ON PROBLEMS ARISING IN THE CLINICAL USE OF CORTISONE, HYDROCORTISONE AND CORTICOTROPIN
Problems Arising in the Treatment of Adrenal Insufficiency
EDMUND B. FLINK, Minneapolis, Moderator
Untoward Effects Upon the Gastrointestinal Tract
ERIC E. WOLLAEGER, Rochester
The Use, Abuse and Limitations of Steroid Therapy in Dermatology
FRANCIS W. LYNCH, Saint Paul
Problems of Dosage in Treatment of Rheumatoid Arthritis
L. EMMERSON WARD, Rochester
Problems Arising From Suppression of Adrenal Cortical Function by Cortisone and Hydrocortisone
ROBERT M. SALASSA, Rochester
Pathology of the Pituitary and Adrenals
WARREN A. BENNETT, Rochester
Question and Answer Period

**Intermission**

10:15 Visit Scientific and Technical Exhibits..Armory

11:00 The Clinical Management of Fulminant Infections in Childhood
EDWARD B. SHAW, Clinical Professor of Pediatrics, University of California Medical School, San Francisco

11:30 ARTHUR H. SANFORD LECTURESHIP IN PATHOLOGY
JOHN R. SCHENKEN, Professor of Pathology, University of Nebraska Medical School, Omaha

P.M.

12:15 Miscellaneous Luncheons
Round-Table Luncheons

1:30 Visit Scientific and Technical Exhibits..Armory

2:00 ULCERATIVE COLITIS
Medical Aspects
J. ARNOLD BARGEN, Rochester
Psychiatric Aspects
L. R. GOWAN, Duluth
Surgical Aspects
CLARENCE DENNIS, New York
Question and Answer Period

**Intermission**

3:15 Visit Scientific and Technical Exhibits..Armory

4:00 SYMPOSIUM ON FRACTURES OF THE SPINE AND LOWER EXTREMITIES

Fractures of the Spine and Pelvis
JOHN C. IVINS, Rochester

Fractures About the Knee Joint
HARRY B. HALL, Minneapolis

Fractures of the Tibia and Fibula in Adults
MALVIN J. NYDAHL, Minneapolis

Fractures of the Lower Extremity in Children
S. S. HOUKOM, Duluth

5:00 Visit Scientific and Technical Exhibits..Armory

7:00 Annual Banquet.....Ballroom, Hotel Duluth

## GENERAL SESSION

**Duluth Armory**

**WEDNESDAY, JUNE 9**

A.M.

8:30 Visit Scientific and Technical Exhibits..Armory

9:00 Diagnosis and Treatment of Hemolytic Anemia
PAUL S. HAGEN, Saint Paul

Anemias in Pregnancy
ROY G. HOLLY, University of Minnesota

Plasma Expanders
A. R. CURRERI, Associate Professor of Surgery, University of Wisconsin Medical School, Madison

Question and Answer Period

**Intermission**

10:15 Visit Scientific and Technical Exhibits..Armory

11:00 Minnesota Division, American Cancer Society

P.M.

12:15 Round-Table Luncheons

1:30 Visit Scientific and Technical Exhibits..Armory

2:00 WHAT'S NEW?

Moderator—F. J. HIRSCHBOECK, Duluth

In Medicine
C. J. WATSON, Professor and Head, Department of Medicine, Medical School, University of Minnesota

In Surgery
OWEN H. WANGENSTEEN, Professor of Surgery and Chairman, Department of Surgery, Medical School, University of Minnesota

In Radiology
LEO G. RIGLER, Professor and Head, Department of Radiology, Medical School, University of Minnesota

In Pediatrics
IRVINE McQUARRIE, Professor and Head, Department of Pediatrics, Medical School, University of Minnesota

In Obstetrics and Gynecology
JOHN L. McKELVEY, Head, Department of Obstetrics and Gynecology, Medical School, University of Minnesota

Question and Answer Period

## SPECIAL SESSIONS

**MONDAY, JUNE 7**

A.M.

10:00 Minnesota Academy of Ophthalmology and Otolaryngology, St. Luke's Hospital Auditorium

Hearing Loss in Children—Detection, Treatment and Rehabilitation.....
KINSEY M. SIMONTON, Rochester

Chronic Middle Ear Suppuration—Today.....
L. R. BOIES, Minneapolis

Retrolental Fibroplasia.....
ERLING HANSEN, Minneapolis

Some Types of Retinal Vascular Occlusions....
HENRY P. WAGENER, Rochester

P.M.

1:00 Round Table Luncheon
Discussion on Vertigo.....
TERRANCE CAWTHORNE, London, England

**TUESDAY, JUNE 8**

A.M.

9:00 Minnesota Society of Clinical Pathologists
Tumor Seminar.....St. Mary's Hospital

Moderator—BENJAMIN CASTLEMAN, Professor of Pathology, Harvard Medical School, Boston, Massachusetts

11:30 Arthur H. Sanford Lectureship in Pathology..
Duluth Armory

P.M.

12:00 Luncheon.....St. Mary's Hospital

2:00 Tumor Seminar (Continued).....
St. Mary's Hospital

6:00 Annual Dinner and Business Meeting

**Plan to attend the annual meeting at Duluth**

**You will learn much that is new and of interest**

# The Dean's Page

## THE DEPARTMENT OF MEDICINE

From time to time on this page, we want to bring you information concerning the teaching and research activities of the various departments and divisions of the Medical School. The Department of Medicine is one of the larger departments and carries an extemely important responsibility for teaching. It is composed of three divisions: Internal Medicine, Dermatology, and Clinical Laboratory Medicine. These divisions are directed, respectively, by Doctors Cecil J. Watson, Henry E. Michelson, and Gerald T. Evans. Dr. Evans is also in charge of the Laboratory Service of the Hospital. The Division of Clinical Laboratory Medicine is responsible for teaching clinical chemistry and microscopy to the sophomore medical students and for teaching the application of laboratory methods to clinical medicine during the junior year.

In addition to the course in clinical chemistry and microscopy, the department is responsible for the following teaching:

1. *Physical Diagnosis,* given in the winter quarter of the sophomore year. Because this course constitutes the student's first contact with patients, it is of particular importance. Students study physical signs at the bedside at the University Hospital and either at the Veterans Hospital, the Minneapolis General, or the Ancker Hospital in St. Paul.

2. *Introduction to Clinical Medicine.* This is a "didactic" course which meets for two hours weekly during the first two quarters and one hour weekly during the third quarter of the Junior year. Students are provided with a lecture syllabus (*The Outlines of Internal Medicine*) which covers the basic material and, for the most part, the class hours are devoted to clinics and demonstrations and discussion of new advances.

3. *The In-Patient Clinical Clerkship* in the Junior year. Students are assigned for ten weeks to the Department of Medicine. Each student spends half of this time at the University Hospital studying patients under close supervision on the wards. Their detailed histories and physical examinations are incorporated into the charts. A student at the University Hospital is responsible also for a considerable amount of the laboratory work. This is carefully supervised. Each student devotes the other half of his medical clerkship to similar clinical work either at the Minneapolis General Hospital or the Veterans Hospital.

4. *The Out-Patient Medical Clerkship* in the Senior year. The student is assigned a new patient presenting for diagnosis and treatment at the Out-Patient Medical Clinic. After taking a careful history and making the physical examination in detail, he sees the patient with one of the staff. After a thorough discussion of the problem and a review of the findings, a decision is reached as to disposition of the patient, i.e., whether the patient should be admitted to the hospital, what further

laboratory or x-ray studies are desired, etc. On the patient's next visit to the Out-Patient Clinic, he will be seen by the same student and staff member who will again attempt to arrive at a definite diagnosis and plan of therapy It may be that a third visit will be required because additional laboratory work or x-ray studies are necessary. Often the patient can be discharged to return to the local physician. In this event, the student prepares the letter to the referring physician, which is reviewed and signed by the staff member. This system provides continuity in follow-up.

In addition, the Department of Medicine is responsible for the admissions service of the hospital. All new patients are worked up either through the system just referred to or, if emergencies, by the medical intern and fellow on call in the admissions room of the hospital.

The staff of the Department represents a number of interests in the broad field of Internal Medicine, and it should be emphasized that every effort is made to sustain the philosophy of internal medicine as a broad discipline in which the patient is regarded as a whole and not from the standpoint of narrow, segregated specialties . There are no specialty services in medicine at the University Hospital. It is true that one of the services in the Variety Club Heart Hospital places more emphasis on cardiovascular disease but students, both undergraduate and graduate, and staff rotate through all services so that a broad integration is maintained.

Research activities in the department are on a rather broad scale. These include studies of brucellosis, staphylococcus infections, chemotherapy, various aspects of liver disease, pigment metabolism, coagulation mechanisms, hypertension (both therapy and fundamental aspects), electrocardiography, metabolic problems, cancer biology, and steroid and mineral metabolism. The fellows in the department are invited to conduct research in collaboration with graduate advisors or with their supervision and advice, either in the areas which have just been mentioned or along some other avenue which is mutually acceptable.

Teaching, service to patients, and research are co-ordinate and interdependent activities in the Department of Medicine as they are in other clinical departments. Each week the department holds a number of conferences and seminars as well as ward rounds. All physicians are cordially invited to attend any of these sessions.

Harold S. Diehl, M.D.
*Dean of the Medical Sciences*

# Medical Economics

**Edited by the Committee on Medical Economics**
**of the**
**Minnesota State Medical Association**
**George Earl, M.D., Chairman**

## ROCHESTER DOCTOR RECEIVES NATIONAL AWARD

Dr. Frank H. Krusen, Rochester, Minnesota, received the Presidential Award of 1953 for his contributions to the employment welfare of the handicapped.

The citation was presented to Dr. Krusen at the annual dinner of the Industrial Health Congress in Louisville, Kentucky, February 24 and 25. Dr. Krusen received the award from Dr. Ross T. McIntire, Washington, president of the President's Committee on Employment of the Physically Handicapped.

In accepting the award, Dr. Krusen said that "proper restoration of handicapped persons depends on three things: (1) appropriate, definitive treatment and physical rehabilitation of the handicapped person in the hospital; (2) proper vocational rehabilitation, and (3) well-established programs for employment of the handicapped." Dr. Krusen is head of the section on physical medicine at the Mayo clinic.

### New Light on Problems

The 14th annual Congress on Industrial Health featured discussions of wide interests to physicians, management representatives and labor leaders. Problems encountered in the maintenance of the health of factory and white-collar workers were topics of interest to those attending. Panel subjects included the stress effects on workers in industry, covering disorders such as neuroses, heart conditions, and ulcers; the effect of prolonged illness occupied another panel subject; the preventive medicine program carried out in some of the duPont plants; industrial implications of emphysema, including the clinical aspects, pathologic physiology, histopathology, radiography and therapy; the difficulties of small plant operators in providing medical care.

### Improvements Noted

Frank G. Dickinson, Ph. D., Chicago, director of the AMA's Bureau of Medical Economic Research, reported that improvements in medical service have greatly increased the economic value of industrial workers. Said Dr. Dickinson:

"In 1900, perhaps one-half of the industrial workers twenty years of age could expect to live to the age of sixty-five. Now, more than four-fifths of them are destined to reach that retirement period.

"The decline in mortality during the working years of life has contributed greatly to economic progress. It has contributed to the solution of some social problems—the probability of becoming an orphan, for instance, has been cut in half."

A joint conference of the Council on Industrial Health and chairmen of state medical society committees on industrial health was held on the day before the opening of the congress. The meeting was sponsored by the American Medical Association's Council on Industrial Health, and physicians and others from the entire country attended to hear of these important phases of industrial health and medicine.

## UNION HEALTH PLANS GET "ONCE-OVER"

A greatly intensified interest in health and welfare plans of labor unions has sprung up within recent years. An excellent example of this interest is seen in a new survey published by the American Medical Association called "Union Health Centers." This survey traces the growth of present plans and notes in the introduction that during and since World War II a much broader and deeper system of such plans has been growing.

It is interesting to note that the pros and cons of such plans have also been presented recently by the *Wall Street Journal*. In a series of articles on the Teamsters Union, the *Journal* states:

"A deep concern for 'health and welfare' has seized leaders of the giant Teamsters Union.

"In the brief period since World War II they've rushed to arrange a bewildering variety of insurance and direct medical service programs."

The article goes on to relate that the interest does not confine itself to the unions themselves, but is manifested in local grand juries and by no less than three Congressional committees. Most of this interest is directed toward finding out about the disposition of certain union funds. One such investigation is being made into the Labor Health Institute in St. Louis, whose finances are being questioned.

According to the *Journal*:

"The institute has its own elaborately equipped five-story clinical building where union members and their families can get their teeth fixed, have allergies treated, their throats sprayed, their bones x-rayed, and buy drugs. It finances service in hospitals, as well. Nearly a million dollars annually is paid in by 175 companies and employer groups whose contracts with the union provide that either 5 per cent or 3½ per cent of their gross payrolls shall go to L.H.I. The larger figure appears in contracts providing full coverage for the dependents of union members."

**No Trouble Elsewhere**

While the *Journal* goes into detail about union leaders being jailed for failure to produce books on health plans, another similar plan, with infinitely different purposes and consequences, has been reviewed. The *Wall Street Journal* itself has featured a story about a mass check-up which is praised by firms, unions and employes in Los Angeles. Called a project in preventive medicine, the plan consisted of one thousand workers at two manufacturing plants being given comprehensive, on-the-job physical checkups that didn't cost them anything. In the *Journal's* words:

"Hoffman Radio Corp.'s television division, with 500 employes, and its neighbor, Leach Relay Co., Inc., with an equal number of workers, offered the health survey as a means of ferreting out prevalent, but often unrecognized, ailments ranging from defective vision to heart trouble or lung cancer."

The company's personnel manager praised the plan: "It's a benefit to the employes, and we believe in the long run it will also benefit the company in terms of better employe relations and reduction of lost time due to illness."

**Medical Support Given**

The idea for the mass employe health screening came from the University of California at Los Angeles, which suggested it to union representatives. It was tried without fanfare and turned out to be successful enough to warrant a thorough test. The *Journal* reports:

"An unusual demonstration of community teamwork by local health organizations kept the actual cash outlay for the survey to $500—or $100 apiece from each of the two companies and the three unions."

Participating organizations and their contributions were: American Cancer Society (pamphlets, and review of some chest x-rays); California Department of Health (equipment for blood tests); Los Angeles City Health Department (equipment and medical advice); Los Angeles County Diabetes Association (equipment, and blood sugar tests at minimum cost); Los Angeles County Heart Association (educational material, electrocardiograms); Los Angeles County Medical Association (co-ordination of projects); Los Angeles County Tuberculosis and Health Association (x-ray equipment, film, technician); University of Southern California Research Laboratory (co-ordination of diabetes tests); University of California at Los Angeles (its Institute of Industrial Relations acted as catalyst in getting companies, unions and other agencies together).

The plan drew favorable comments from all of those participating, union officials, company officials, agency officials and employes themselves. The plan was a sample of what can be done if all concerned work together to promote a worthy project for the good of all. Those who worked out the project feel that if used in other areas, care should be taken to see that all agencies are in on it and that all have a part in planning and operation.

## COMMISSION BRINGS RECOMMENDATIONS

The Commission on Financing Hospital Care has recently published its recommendations, after two years of intensive study and investigation. The thirty-four member commission completed its study with contributions amounting to $556,000 from the Blue Cross commission, Health Information Foundation, John Hancock Mutual Life Insurance company, W. K. Kellogg Founda-

tion, Michigan Medical service, Milbank Memorial Fund, the National Foundation for Infantile Paralysis and the Rockefeller Foundation.

Pointing out that in eight states three-fourths of the population are participating in voluntary prepayment plans for defraying hospital expenses, the Commission states:

"The financing of hospital care and the maintenance of standards of hospital service will depend increasingly upon maximum coverage of the population with voluntary prepayment at the lowest possible cost compatible with the inclusion of adequate benefit provisions."

The Commission report then goes on to make the following recommendations:

1. Hospitals should take the initiative in establishing local study committees to evaluate all prepayment plans in their area. Recommendations for improvement should be made to the prepayment agencies, the hospitals and the public.

2. Prepayment agencies should take the initiative in mobilizing community leadership to inform the public concerning the frequency of hospitalized illness, the rôle of voluntary prepayment in meeting costs, factors affecting cost, and benefit provisions.

3. Methods should be developed to enroll the following who are not presently covered by prepayment in sufficient number: dependents of present contract holders such as infants, children over 18, parents and other relatives; the self-employed; farm operators and employes; employes of small firms; employes who retire under pension plans; employes temporarily without employment because of short layoffs or change of employers; domestic employes; migratory workers; persons receiving workmen's compensation benefits.

4. Employer participation in costs of prepaid protection should be encouraged.

5. Methods should be explored for the coverage of individuals and families by prepayment during periods of temporary unemployment.

6. Methods should be explored for continuation of coverage during periods of prolonged disability.

7. Programs might be developed for coverage of individuals and families who are without income from employment.

8. Low-income families should be encouraged to budget through prepayment for the cost of adequate protection. Experimental programs should be instituted to reduce the cost of prepayment to bring protection within reach of this group.

9. Hospitals, prepayment agencies and community groups should support, promote and interpret administrative practices in the operation of prepayment which help attain maximum coverage.

10. Hospitals and community groups should aid in enrollment policies and techniques to permit those not covered to attain prepayment protection.

11. Hospitals should help conduct area-wide studies of prevailing benefit provisions and their effect upon the financing of hospital care. Recommendations should then be made to the public regarding basic benefit provisions required for adequate protection of public and hospitals.

12. Hospitals, co-operating with others, should assume leadership in developing measures and educational programs designed to assure most efficient and economical use of prepayment funds. Consideration should be given to efficiency in provision of hospital services, unnecessary use of hospital services, appropriate use of hospital services and efficiency in operation of the prepayment agency.

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## OFFICE HEMATOLOGY

*(Continued from Page 279)*

The majority of stain trouble is *buffer* trouble. We use the following buffer adjusted to a pH of 6.4: potassium phosphate, monobasic, 6.63 gm.; sodium phosphate, dibasic, anhydrous, 2.56 gm.; distilled water to 1,000 cc. Check the pH of the buffer frequently with several crystals of hematoxylin in a test tube of buffer solution. A pink-lavender color develops in two minutes if the buffer is satisfactory. Red-purple means it is too alkaline; yellow means it is too acid. Adjust with weak acid or bicarbonate as indicated. The stain-buffer times varies with each new batch of stain. A good stain is pink on the slide. Erythrocytes should be light buff; the nuclei are blue-purple and are in sharp contrast to the cytoplasm. Eosinophilic granules are bright red and neutrophilic granules are pink to lavender.

No degree of diagnostic acumen can correct for a poor smear or a poor stain. When sending blood smears for consultation, send one stained and one unstained smear with pertinent clinical and hematologic data. Give the age of the patient. Bone-marrow preparations require exacting and special technique and are not office procedures. It's not "old fashioned" to look at your own blood smears. You cannot afford to ignore them!

# Minneapolis Surgical Society

**Meeting of December 3, 1953**

**The Vice President, Maynard Nelson, M.D., in the Chair**

## SPONTANEOUS RUPTURE OF THE LIVER

### A Case Report with Recovery

**L. A. STELTER, M.D., and G. L. PETERSEN, M.D.**
**Minneapolis, Minnesota**

THE FIRST known case of spontaneous rupture of the liver was reported by Vesalius at some undetermined date. Andral, in 1829, sketchily reported two cases with complicating lesions which may have been gummata or carcinoma. However, in many of these earliest cases, there is some probability that gastric hemorrhage, secondary to liver disease, may have been mistaken for actual rupture of the liver. According to Devic and Beriel and later Mazel, the following theory had been formulated: "In traumatic rupture, the rupture is the cause of hemorrhage. In spontaneous rupture, the hemorrhage is the cause of rupture."

Rupture of the liver is a result of spontaneous tearing or trauma. The chief traumatic causes are: (1) blunt force, (2) open injuries, such as stab wounds, gunshot wounds and war injuries.

Of all the organs in the abdominal cavity, the liver is the most likely to be injured when the abdomen is hit in any part by blunt force. Street and automobile accidents; falls from scaffolding; burying accidents; war injuries and hoof beats are the main causes. In 494 postmortem examinations of rupture of abdominal organs through blunt force, liver rupture rates first with 59.9 per cent. The right lobe has been found ruptured much more frequently than the left. The relationship is about 65 to 35.

Spontaneous rupture of the liver is due to enlargement of the liver from disease within stretching Glisson's capsule, which has a certain degree of tension. The chief diseases which may cause the liver to burst are fatty degeneration, malaria, tuberculosis, syphilis and malignancy. It occurs when the liver is more friable and during digestion when it becomes hyperemic and enlarges.

The greatest danger in liver rupture is hemorrhage. In fact, this is the main cause of a fatal outcome. The large intrahepatic veins have very thin walls, no valves are present, and there is no retractibility, therefore the bleeding is persistent and spontaneous control is rare. Because of the large venous blood supply in the liver, the hemorrhage is more likely to be of venous nature and enormous amounts of blood may be found in the abdominal cavity.

Extensive liver injuries have a very high mortality rate and statistics from the war give a lower mortality rate (60 to 80 per cent), which may be erroneous, as a considerable number die immediately on the battle field because of massive hemorrhage.

Clinical signs of ruptured liver and differential diagnosis are: (1) symptoms of general shock, (2) general symptoms of abdominal injuries, (3) special symptoms caused by the injured liver.

*Shock.* The patient lies still with an expression of fear in the face. The skin is pale and clammy; the mind is clear, but mental activity is diminished. The respiration is shallow and frequent, deep sighs interrupting normal respiration. The pulse at first is full and bounding, later thready and rapid, and the blood pressure progressively falls.

*Special Signs of Liver Injury.* When loss of blood occurs in the abdominal cavity, peritoneal irritation occurs immediately, the intensity depending on the rapidity of intra-abdominal hemorrhage. Pain after rupture of the liver, is quite characteristic. It is especially felt when pressing the finger into the intercostal space. Pain radiates downward and backward and also into the right shoulder. This referred pain is due to diaphragmatic irritation through the phrenic nerve and into the cutaneous branches of the dorsal scapular nerve. Breathing is shallow, more fre-

TABLE I. CAUSES OF RUPTURE OF THE LIVER OTHER THAN VIOLENT MUSCULAR EFFORT*
*From Annals of Surgery, 118: 397, (Sept.) 1943.

| Date | Author | Age | Cases | Operation | Cause | Result |
|---|---|---|---|---|---|---|
| 1829 | Audral | | 2 | Autopsy | Carcinoma or gumma | Died |
| 1831 | Honore | | 1 | Autopsy | Multiple caries | Died |
| 1836 | Gerard | 52 | 1 | Autopsy | None | Died |
| 1844 | Saequepec | 44 | 1 | Autopsy | Syphilis | Died |
| 1847 | Fauconneau & Dufrense | | 1 | Autopsy | Typhoid fever | Died |
| 1849 | Abercrombie | 35 | 1 | Autopsy | Pregnancy | Died |
| 1864 | Heinzdman | | 2 | Autopsy | Tuberculon Peritonitis | Died |
| 1884 | Chrari | 40 | 1 | Autopsy | Cancer | Died |
| 1886 | Heinzdman | 18 | 1 | Autopsy | Pneumonia | Died |
| 1890 | Laureneau | 16 | 1 | Autopsy | Syphilis | Died |
| 1892 | Pauly | 47 | 1 | Autopsy | Biliary colic | Died |
| 1892 | Poljakoff | | 1 | Autopsy | Epilepsy | Died |
| 1898 | Bar & Reim & Neubert | | 2 | Autopsy | Syphilis | Died |
| 1899 | Carnabel | 26 | 1 | Autopsy | Osteitis | Died |
| 1902 | Bernard | 62 | 1 | Autopsy | Hemiplegia | Died |
| 1905 | Nauwerck | | 1 | Autopsy | Common duct stone | Unknown |
| 1906 | Derri & Beriel | 41 | 1 | Autopsy | Syphilis | Died |
| 1921 | Walz | | 1 | Autopsy | Aneurysm | Died |
| 1932 | Rethon & Cadein | 34 | 1 | Operation | Exercise & cough | Recovered |
| 1933 | Mast & Streamer | | 1 | Autopsy | Pneumonia: Ca. of liver | Died |
| 1934 | Robin | 28 | 1 | Operation | Trauma | Recovered |
| 1936 | Bradley & Garrett | 3 | 1 | Operation | None | Recovered |
| 1937 | Sciacia | | 1 | Autopsy | Toxic infection | Died |
| 1938 | McEwan | 8 | 1 | Operation | Malaria | Died |
| 1940 | Coniden | 9 | 1 | Operation | Slight trauma | Recovered |
| 1943 | Rademaker | 32 | 1 | Exploratory | Pregnancy | Recovered |
| 1946 | Links | 42 | 1 | Operation | Pregnancy | Recovered |
| 1946 | Sanes and Kaminiski | | 1 | Autopsy | Pregnancy and eclampsia | Died |
| 1951 | Haller, Abels & Straus | 34 | 1 | Autopsy | Eclampsia | Died |
| 1953 | Wright and Ball | 61 | 1 | Operation | Hypertension | Recovered |

quent, and may be painful. Dullness in the right side of the abdomen gradually increases and spreads downward as the blood flows down the right abdominal gutter. The dullness may shift if the patient is turned to the left. Rectal or vaginal or rectovaginal examination reveals fullness in the pelvis and tenderness. Bradycardia may last longer than in other types of intra-abdominal hemorrhage and this is due to resorption of bile from the abdominal cavity. Bile salts have a depressing influence on the heart. Diagnosis of bile salts may be made by tapping the abdominal cavity with a No. 20 spinal needle; a procedure which I have done on various occasions of abdominal injury or suspected ectopic pregnancy. No harm will be done to the small or large bowel where the abdomen is free from adhesions. The examination of the blood for bile and bile salts and for liver cells will be helpful. X-ray examinations, upright and flat abdomen, will only help in ruling out air under the diaphragm in rupture of gas containing viscera. Auscultation reveals a near silent abdomen.

*Complications.* (1) death from hemorrhage, (2) cholascos (effusion of bile into the abdominal cavity) causing bile peritonitis, (3) permanent biliary fistula, (4) infection of the liver wound, (5) embolism of liver tissue, (6) infarction of the liver, (7) lung complication—pneumonia (due to emboli), (8) hepatorenal syndrome (nephrosis of the kidneys), (9) eye symptoms, simulating albuminuria retinitis.

*Treatment.* From the statistics of Elder, Terrier, Auvray and others, who have shown that the mortality in non-operated cases was as high as 85.9 per cent, while in the past decade, with modern surgical intervention, the mortality has been reduced from 53.8 per cent to 28.5 per cent, we feel that this is an acute urgent surgical entity. The patient should be transfused by multiple portals of blood and operated upon immediately.

## Case Report

The patient, a white female teacher, aged twenty-three, a student at summer school, gave the following history: While eating her Sunday dinner with a friend at 2:00 P.M., she was suddenly stricken with an acute pain in the right shoulder. She was unable to finish her meal and had to be assisted to a lounge where she lay for a short time having a lady massage her shoulder because of the pain. She then experienced pain in the upper abdomen—severe and intense—vomited her dinner and retched several times, after which she felt relieved for about an hour. During this time she was taken to her room, at a Girls' club, and went to bed, complaining to her roommate that her abdomen was getting more and more painful, that she felt faint and weak, wanted to vomit, and that she had a severe backache.

I saw the patient at 5 o'clock p.m., a tall, fairly well-developed female, holding her hand on her upper abdomen. She was somewhat pale, the examination revealed she had a normal pulse, blood pressure was 70/30, heart and lungs were normal, abdomen was held tense and with boardlike rigidity above the navel and across to the right lower quadrant. Vaginal examination was negative. She was sent to Fairview Hospital by ambulance and upright and flat x-rays revealed no air under

the diaphragm. Hemoglobin—70 per cent; R.B.C.—3,700,000; W.B.C.—16,000; P.M.N.'S—94 per cent; lymphocytes—6 per cent. Urinalysis revealed sugar—four plus also acetone and diacetic acid. There were 40-50 R.B.C.s and pus cells—20-30 per high powerfield pus cells. Re-examination of her abdomen at 8 o'clock p.m. by Dr. Petersen and myself, revealed a benign abdomen. There was no rebound tenderness and no rigidity at this time. The patient laughingly remarked, "Why do I have to stay in the hospital? My pain all left me when I came here in the ambulance."

We decided to treat the patient conservatively on the basis of a diagnosis of sealed perforated posterior duodenal ulcer, complicated by a severe diabetes and pyelitis. Nasal suction was instituted and Dr. Petersen treated the diabetes by I.V. glucose and large doses of insulin. Re-counts of laboratory work were ordered for the following morning. Her blood pressure and pulse were observed at hourly intervals during the night and no change was noted. At 7:30 a.m. the next morning no change was noted except for some abdominal distention. On rectal examination some fullness was felt in the lower pelvis. Re-check of x-rays and I.V. pyelogram were ordered.

At 11 o'clock a.m. an acute change in her condition was apparent. The pulse became rapid. She had become pale and clammy. The vaginal examination, at this time, revealed marked tenderness. I then made a diagnosis of acute abdominal catastrophe, as the laboratory at this time, reported that her hemoglobin had dropped to 55 per cent and the red blood cell count was 2,500,000. A diagnosis of intra-abdominal hemorrhage was evident. An emergency operation was performed.

Under spinal anesthesia, with two units of bloods and normal saline running in the veins, a "peak" right paramedian incision was made. On finding the abdomen full of blood, the incision was continued down to the pelvis and both tubes and ovaries explored and found normal. Exploring the right side and following up to the liver, I found that the girl had a ruptured right lobe of the liver. The incision was extended to the xyphoid and the right rectus muscle cut across to the right costal margin, the blood evacuated by suction, approximately 1 quart, and a rent, the size of my hand, was found in the right lobe, extending up to the dome of the liver. The entire surface of the right lobe was dark from subscapsular hemorrhage and echymosis. The active bleeding, which was profuse from the liver rent, was then controlled by large gauze packs. These were then gradually removed after the patient's pulse and blood pressure stabilized and replaced by oxycel and gel foam, seven such packs were used to fill the rent and the liver was then sutured with mattress sutures. This, however, proved very difficult because of the friability of the organ. Two large cigarette drains were placed between the chest wall and the liver rent and the abdomen was then closed, the peritoneum with chromic 0 doubled, the fascia with through and through wire and cotton. A third transfusion was given after surgery and her immediate postoperative condition was good. Her postoperative progress was satisfactory and her diabetic condition was controlled by I.V. fluids and insulin under Dr. Petersen's supervision. Nasal suction was continued.

A liver biopsy, at the time of surgery, was considered and would have been helpful, but in view of the critical condition of the patient, and the black, discolored liver tissue, I felt the procedure would only add insult to an already badly injured organ.

The remaining postoperative condition of the patient progressed satisfactorily. After the third day, the nasal suction was removed. One drain was removed on the fourth day and the second on the eighth day. The patient left the hospital on the thirteenth day postoperatively and was seen subsequently in the office.

A letter from her on the second month after surgery, reported her to be in fine physical condition.

## Comment

In 1943, Rademaker collected twenty-nine cases of spontaneous rupture of the liver, including the case of Vesalius. Since then four cases have been added, three associated with pregnancy and one with hypertension.

Because a biopsy of the liver was not made at the time of surgery, I believe one is justified in assuming that this case of spontaneous rupture was due to some metabolic disturbance of the liver, associated with a mild diabetes.

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# Minnesota Department of Health

## GAMMA GLOBULIN (IMMUNE SERUM GLOBULIN)

### For Poliomyelitis

Gamma globulin is available for direct intimate contacts, forty years of age and under, of reported cases of clinical poliomyelitis on receipt of the name, age and weight of the contact, and identification of the case of polio. The dose of globulin for poliomyelitis contacts is 0.14 cc of globulin per pound of body weight, to a maximum of 20.0 cc and should be given as early as possible following exposure. It is probably of little value if *ten* days or more have elapsed after exposure.

### For Infectious Hepatitis

Gamma globulin is regularly available for close and continued contacts of cases of infectious hepatitis (jaundice). Since the product is of no value once symptoms of the disease have begun, and even late in the incubation period, this means that the product should be used largely in household contacts and those living in small groups where the contact is essentially the same. The dose of globulin for contacts of infectious hepatitis depends on the body weight and is 0.02 cc of globulin per pound of body weight, with a maximum of 2.0 cc for any one individual. Globulin should be given to all household contacts who do not give a history of having had the disease, including adults.

### For Measles

Gamma globulin for measles has been available for a number of years and most physicians are familiar with its use for this purpose. Those at greatest risk are children three and under. As a general rule, the amount given is aimed at modification of the disease and the development of permanent immunity by a mild attack. Occasionally it is wise to aim at complete prevention, even though no permanent immunity results. Only contacts who have had a recent definite exposure should receive globulin. Those who have had measles already should not be inoculated.

For modification, the dose of globulin for children definitely exposed to measles is 0.02 cc of globulin per pound of body weight. When given during the first six days following exposure, this amount will usually produce a modified case of measles. If complete prevention is urgent, the dose is five times as large. Gamma globulin should not be given if ten days or more have elapsed after exposure, since its value at that late date is negligible.

### For Other Conditions

Globulin is not distributed for other conditions for which its value has not been established. This includes mumps, german measles, diarrhea, or for treatment of symptoms once a disease has developed.

### Reporting of Cases

Since the Department's supply of gamma globulin is allocated on the basis of numbers of reported cases, it is important that all cases of the above diseases be reported to the State Board of Health as required by regulation.

## LABORATORY EXAMINATION OF GASTRIC WASHINGS FOR TUBERCLE BACILLI

Increasing numbers of patients with tuberculosis are receiving post sanatorium out-patient chemotherapy, extending over long periods. It is important that such patients have careful evaluation of their progress at intervals of three months or so, including x-rays, laboratory studies, and tests for the presence of tubercle bacilli. Usually the latter tests are performed by culturing the gastric washings of the patient.

When gastric washings are collected for culture for tubercle bacilli, the specimen should be immediately put into the special bottle prepared for this purpose. This bottle contains a buffering chemical which neutralizes the acid in stomach washings and inhibits the detrimental action of the gastric acidity on the tubercle bacilli. Unless this is done, the bacilli are soon killed and cultures will show no growth.

The Minnesota Department of Health furnishes these special outfits for the mailing in of gastric washings for culture and urges that the special outfit be used whenever there is to be delay between the collection of the specimen and its inoculation onto culture media in the laboratory.

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## FATIGUE AND HEALTH

There seems to be little doubt that physical and intellectual fatigue, as well as painful emotions, decrease resistance of the body to infection; but the physiologic mechanisms which determine these important relationships are totally unknown. Despite all this ignorance, however, a belief shines as a beacon to guide our efforts. It is that the defense mechanisms of the body which promote resistance to infection appear to be closely related to the physiologic processes which make for good health—good health being here measured in the terms broadly referred to as well-being by the layman. The inner logic of the program of antituberculosis associations should lead them to become truly health associations. For it can hardly be doubted that, in final analysis, health is a positive attribute rather than merely the absence of disease.—Rene J. Dubos, Ph.D., American Review of Tuberculosis, July, 1953.

# History of Medicine In Minnesota

## MEDICAL HISTORY OF THE COUNTY OF YELLOW MEDICINE

MILDRED B. LEE
Granite Falls, Minnesota

*(Continued from March, 1954, issue)*

**William Moffat Stratton** was born in St. Lawrence County, New York, in 1844. His education began in the common schools of that state, and continued in the schools of Wisconsin when the Stratton family moved west in 1854. He received his medical training at Ann Arbor, Michigan, and at Chicago Medical College which is now a part of Northwestern University. He graduated from the latter school in 1869.

Soon after graduation Dr. Stratton began practice at Ortonville, Minnesota. In 1873 he moved to Minnesota Falls, which was a new settlement on the south bank of the Minnesota River just above Minnesota Falls in Yellow Medicine County. Here he was joined by another physician, Dr. E. N. Falk, and although the two doctors did not practice as partners they were partners in a drug business which they established and carried on for several years.

Drs. Stratton and Falk were both graduate pharmacists as well as physicians.

When Dr. Stratton came to Minnesota Falls the little town was competing for the honor of being named the county seat, and it had made astonishing progress in the short time it had been in existence, boasting a flour mill, hotel, blacksmith shop, two stores, a law office, and several saloons. A newspaper would add to the town's prestige, and Drs. Stratton and Falk became partners in establishing the first newspaper in Minnesota Falls and in the County, the *Minnesota Sentinel.* The first issue appeared October 1, 1874.

The paper was a modest effort; it was a six-column folio with the outer pages filled with local advertisements, some in Norwegian, professional cards of the doctors and lawyers, legal notices, and local news items. The few copies which are on file in the office of the clerk of court contain tax lists, and also a fairly full account of a libel action brought by Dr. N. M. Smith against Editor Hogue of the *Granite Rock.* In 1876 the *Sentinel* was sold to a Mr. Bennet who kept it in publication for another two years, after which it died from lack of support.

When Granite Falls became the county seat the population of Minnesota Falls began to move away from the town. Dr. Stratton moved to Granite Falls about 1877 or 1878, and became a member of the first town council in 1879. In 1881 he became president of the council, and later served as Health Officer of the city and as a member of the Board of Education. In the latter capacity he was one of the board members who chose the site for a school in West Granite Falls, and brought about the erection of the first school building on the present school grounds.

Dr. Stratton married Adeline Sarah Enos, whose father was a carpenter and built many houses in Granite Falls. One of these, Number 235, on Tenth Avenue,

is now the home of Paul D. Stratton. Dr. Stratton himself lived in a house on south Prentice Street which is still standing, though somewhat altered, as No. 1165 Prentice.

When he first came to Granite Falls, Dr. Stratton became a partner of Dr. Cyrus P. Fuller who was the first physician to locate in Granite Falls.

Dr. and Mrs. Stratton were kindly, generous people who became popular residents of the village. In youth they were considered a rather handsome couple. Dr. Stratton was of average height, having a rather round face with large and slightly protruding eyes, a full lipped and usually smiling mouth. He walked with a slightly shuffling gait and always carried an umbrella—his own or someone else's; and it was usually in varying states of dilapidation; he liked to have something to carry as he walked along. Usually he walked with down-bent head and his manner was quiet and kindly. He had a keen sense of humor. In temperament he was easy-going; although he kept an extensive set of books he never pressed anyone for payment, and indeed seemed to be indifferent not only to obligations owed to him but to those he himself owed. He was devoted to his patients. He would answer calls at any time of day or night and give freely of his time and effort. Sometimes he suffered keenly over the death of a patient. He was a physician and surgeon of exceptional ability, and neither weariness nor other circumstance ever seemed to affect his skill and judgment in reference to medical practice. Educated in a time when the germ theory was unknown he never adapted himself to new techniques of sterilization and sanitation.

Dr. Stratton practiced under most primitive conditions. The doctor's offices had none of the conveniences essential to comfort. A wood burning stove furnished heat, water was carried in, illumination was furnished by coal oil lamps; and unless the doctor was lucky enough to be able to employ help the doctor did what janitor work was required. Like other pioneers he got around the country as best he could; by horseback, with horse and buggy, sleigh or cutter. For many years Dr. Stratton and Dr. Flynn of Redwood Falls were the best known, if not the only doctors in a very large territory, and they were on the road day and night. At one time Dr. Stratton owned seven horses and it is remembered that Mr. Charlie Rice drove for him. A driver was a necessity not only to share the labor of getting the buggy through mudholes or snow banks but to permit the doctor to rest between calls.

When the railroad came to Granite Falls in 1878 Dr. Stratton was appointed the first surgeon and physician of the road at that point, and he held this post until his death, so that he was then the oldest physician of the line in point of service. Soon he began to travel by train to the more distant points to which he was called; the *Granite Falls Journal* mentions that "Dr. Stratton is on wheels most of the time," attending to his practice, and meeting in consultation with other physicians.

The Stratton home was a favorite meeting place for the children of the neighborhood. Dr. and Mrs. Stratton had a family of their own: three sons, Frank E., William, and Paul D., and three daughters, Adeline E., Edna, and Susie. Susie loved to help her father; she is remembered as a little girl with braids down her back and glasses on her nose, using the vaccinating needle like a veteran upon the children who were candidates for this rite. But it was Edna, not Susie, who became a nurse.

The Strattons had their sorrows; the little girl, Adeline, died in 1894; the doctor's health suffered and he was away for five months for treatment and rest. Younger doctors with more modern methods and techniques settled in Granite Falls and other parts of the county which he formerly served, cutting into his

practice; and in his later years the doctor had less strength for and interest in the arduous duties of the country practitioner. Throughout his career he was respected for his exceptional skill and medical knowledge, for he often succeeded where other physicians failed. His personality endeared him to all who knew him; he brought to his patients the assurance of his sincere interest and concern.

When plans were made for the erection of a tubercular sanatorium at Granite Falls for the tubercular patients of three countties, Dr. Stratton was chosen as a member of the board from Yellow Medicine County, and this position he held during the time the buildings were being erected and the first patients received at the new sanatorium. He was also a member of the Camp Release Medical Society and of the State Medical association.

Mrs. Stratton died January 11, 1912, after a rather long period of declining health. Dr. Stratton died November 10, 1916, of a stroke of apoplexy, at the age of seventy-two years. He was survived by his sons, Frank E., William, and Paul D., his daughters, Edna Maercklein, and Susie (Mrs. M. H.) Geehan. Edna Maercklein was the wife of a doctor and assisted in the operation of a hospital at Oakes, North Dakota, at one time.

Present survivors of the Stratton family are Paul D. Stratton, an attorney who has practiced at Granite Falls for about forty years; Susie, who was Mrs. M. H. Geehan of Graceville, Minnesota, was survived by a son, Robert Geehan, who is a mining engineer, and a daughter Adeline who is now employed by the city of Minneapolis.

### Albert R. Torgersen

"Dr. A. Torgersen of Fargo, North Dakota, is stopping at E. P. LeSeuer's Drug Store. He has had extensive experience and will gladly attend any demands for his professional services."

This announcement in the issue of the *Canby News* of October 9, 1885, heralded the arrival of Dr. Albert R. Torgersen, who was to spend many years in the practice of medicine in Yellow Medicine County.

Albert R. Torgersen was born in Oslo, Norway, and received his education and medical training in the schools and Universities there. He came to the United States as a young man and settled first in Wisconsin. From Wisconsin he came to Minnesota, locating in Alexandria, where he met the young woman who became his wife, Miss Addie Corneliuson, who was also of Norwegian descent. From Alexandria he went to Fargo, North Dakota, leaving that city for Canby in 1885.

When Dr. Torgersen came to Canby he had two children, Rudolph Charles, who was born in Alexandria, and William Cornelius, who was born at Fargo, North Dakota. In appearance the doctor was small in stature, dark, always well groomed and giving an impression of poise and good breeding. He wore a large mustache, and in disposition was kindly, but very outspoken and so strong in his convictions that he was sometimes regarded as "contrary."

Dr. Torgersen seemed to have trouble establishing himself in Canby. A rumor to the effect that he was not qualified to practice in the state caused at least one patient to refuse to pay for his services, and Dr. Torgersen promptly sued for his fees. Thereafter he announced through the *Canby News* that he had been educated in Norway and had complied with the requirements of state law and was licensed to practice in Minnesota.

Dr. Torgersen was also a graduate pharmacist, and after three years in Clarkfield, during which time another son, Roy Jay, had been born, the doctor decided to go into the drug business in connection with his practice. In 1888 he purchased the stock of the Clarkfield Drug Store and on September 20, the *Canby News*

announced that "Dr. Torgersen has taken possession of the drug store he recently purchased at Clarkfield. He will probably do well in his new venture and the outlook is flattering." As to what Clarkfield was like at this time, there is this comment:

"Clarkfield is a new town started upon Monson's place two years ago and now numbers 300 inhabitants. It is a great wheat market for a town of its size and has taken in as high as 5,000 bushels of wheat a day. Yellow Medicine must be a wheat growing county, one 20 acre piece yielding 900 bushels of Blue Stem wheat."

The little village was in the heart of a rich farming country and its population was predominantly Norwegian. Dr. Torgersen was the first doctor to locate in Clarkfield.

The doctor's first offices in Clarkfield were in the old Myhre Building, and his family lived in the rooms adjoining them. Shortly after the arrival in Clarkfield another son, Cleve Reuben, was added to the family circle. The doctor was a Democrat and a great admirer of Grover Cleveland, and Cleve Reuben was named for him; but during Cleveland's administration the doctor suffered heavy financial losses, as did many other business and professional men, in the panic of 1893.

In 1888 Dr. Torgersen was elected county coroner; and he was very conscientious in exercising the duties of this office, as shown by the case of Peter Strand. In this case the dead man was found in a meadow where the body had evidently lain for some time undiscovered, despite the fact that one Luders had been mowing in full view of the body for some days. A quantity of blood, or evidences of blood, was found, and there were wounds in the throat. But the man was buried in spite of protests, as he was an immigrant and without friends, but believed to have had money as he contemplated buying a farm. Evidently public opinion compelled the authorities to take further steps, so the body was exhumed and an examination made. The *Canby News* reported that the doctor felt he had "been hindered in his examination," and believed that murder had been done, but the authorities refused to take action. The county attorney was A. J. Volstead.

Dr. Torgersen also served as County Physician for the Clarkfield medical district.

The fifth child born to the Torgersens was Pearl Celia, and when the little girl was still very young her mother died very suddenly.

After this bereavement the doctor was left with the responsibility of caring for the children in addition to his medical practice and drug business. Nevertheless he managed; the family lived in a home in the west part of Clarkfield where the doctor had an opportunity to indulge his hobby of gardening. During the summer his vegetable garden and gorgeous flower beds made his home a show place. He also found time for crayon work and drawing which showed his considerable talent as an artist.

The rewards of medical practice in this small community were small. The usual charge for a confinement case was $5.00; a country call was made for $2.00, and a dollar had to be paid out for a livery rig. Then often the farmer would tell some story of hard luck, and Dr. Torgersen would tell him just to forget about the fee. People respected Dr. Torgersen's ability as a doctor, and although new and younger doctors came to the community there were many people who thought no one could do quite as much for them as Dr. Torgersen.

The doctor's son Rudolph Charles answered the call for volunteers in the Spanish American War, and shortly after his discharge from service died in Seattle, Washington.

Another boy to go into the military service was Cleve Reuben, who served in World War I, and was killed in action.

In 1917 Dr. Torgersen gave up the practice of medicine in Clarkfield and went with his son Roy Jay to Hawarden, Saskatchewan, Canada, and lived there until his death.

He was survived by his son William Cornelius, who is now in business in Starbuck, Minnesota; Roy Jay who is in business in Winnipeg, Canada, and Pearl Celia (Mrs. A. G. Drysdale) who resides in Arcadia, California.

**William B. Torgerson** was born in Worth County, Iowa, on October 27, 1872. He was the son of Rev. Torger A. Torgerson and Dina Anderson Torgerson, and was christened Wilhelm B. Torgerson. He received his early education in the schools of Iowa, and then entered the College of Homeopathic Medicine and Surgery of the University of Minnesota. He graduated in 1897, and began his practice in Cannon Falls, Minnesota, the same year.

In November, 1908, Dr. Torgerson located in Clarkfield. The *Cannon Falls Beacon* in commenting on Dr. Torgerson's departure stated:

"Dr. Torgerson's departure will be greatly regretted. He is a thorough gentleman and a skilled physician who has gained many friends during his stay in Cannon Falls."

In Clarkfield Dr. Torgerson occupied offices over the Clarkfield State Bank, with living quarters in connection so that he could be conveniently "found day and night" as stated in the *Clarkfield Advocate*. He was a tall, slender, dignified young man, with brown hair and eyes, a small well trimmed goatee, and is generally described as "aristocratic looking."

In 1899 Dr. Torgerson was appointed Health Officer for the village of Clarkfield.

In June, 1900, Dr. Torgerson took a brief vacation, during which, on June 14, he was married to Miss Ingeborg Johanna Ohnstad of Watsedo, Minnesota; ("the bride is one of Goodhue's fairest and most accomplished daughters") and the young couple returned from a wedding trip to take up housekeeping in the apartment formerly used as offices and living quarters, while the doctor settled in new offices over the bank. According to the *Advocate*, Dr. Torgerson "came here about two years ago, and by strict attention to his profession has built up a good practice and enjoyed a large degree of popularity in social circles."

Throughout his life Dr. Torgerson was known as William B. Torgerson rather than by his baptismal name of Wilhelm.

The first child born to the Torgersons was a girl, born May 14, 1901, and named Dora. A son, Erling A., was born October 28, 1902, Harold W., born March 27, 1905, and Theodore A., born May 22, 1907.

During the period of residence in Clarkfield the doctor and his wife were very well liked, and he enjoyed a good medical practice. He also occupied for a time the position of postmaster of Clarkfield.

In 1907 Dr. Torgerson removed to Greenbush, Minnesota, and practiced there until 1912. Another son, William B., Jr., was born there on February 12, 1909. In 1912 Dr. Torgerson moved to Oklee, Minnesota, and practiced there until his death.

Dr. Torgerson took an active part in public affairs in Oklee, and had many interests in addition to medicine. He went into banking, became a member of the school board, the secretary of the creamery at Oklee, and was active in church matters.

At the time of his death Dr. Torgerson was one of the oldest medical practitioners

in the state. He was survived by his wife and five children. Mrs. Torgerson continues to live at Oklee, Minnesota; daughter, Dora, is Mrs. Robert E. Bilbrough of Overland Park, Kansas; Erling A. Torgerson lives at Thief River Falls where he is in business; Harold W. Torgerson lives in Evanston, Illinois, and Theodore is at Annapolis, Maryland, while William B. Torgerson resides in Oakland, California.

Dr. Torgerson was a member of the Evangelical Lutheran Church. A brother, Rev. Abel Anderson, lived at Montevideo and the Torgerson children were baptized by him and all became members of the "Synod" group of the Lutheran church.

Dr. Torgerson died July 7, 1952, of Parkinson's disease, for which ailment he was hospitalized for three years previous to his death.

**Henry G. Townsend** was born in Maine in 1823. He came west and it is believed that he lived for a time in Chippewa County, Wisconsin, for that was the place of residence of his divorced wife, Alice, and his son, Edgar H. Townsend.

Dr. Townsend came to Granite Falls in 1877, at the age of fifty-four years. He established himself in a house in East Granite Falls, Chippewa County, and inserted his professional card in the *Granite Falls Journal.* The issue of that paper on July 25, 1877, introduced him as a

"Homeopathic Physician, Surgeon and Obstetrician. Special attention given to cronice (sic) diseases and diseases of women and children. Office and residence on east side opposite E. J. Fitch's Hotel."

This places him on the north side of what is now Baldwin Avenue near the Milwaukee depot.

Dr. Townsend was a quiet, elderly man; he was a member of the first library association of Granite Falls.

In 1882 Dr. Townsend had moved to Canby, where he had an office "1 door south of P. O." His professional card stated that "He will hold himself in readiness to attend calls at all hours night and day." Dr. Townsend is also said to have practiced in the small settlements near Canby more extensively than in the city of Canby itself.

On January 26, 1883, he was stricken with a heart attack and died at the age of sixty years. Probate proceedings of the county show that his only heir was his son, Edgar H. Townsend, sixteen years old, a resident of Chippewa County, Wisconsin.

**M. N. Triplett** was graduated from the University of Louisville in 1895, and was licensed in Minnesota January 11, 1898.

He was born in one of the southern states, either Tennessee or Kentucky, and educated in the schools of the South. He always kept his southern manner of speech, which was quite distinctive. He was a fine looking young man, of medium height and coloring, and good physique.

Dr. Triplett located in Hanley Falls shortly after his graduation and practiced there two years. At the end of that time he went to Dawson, where he built up a good practice and was regarded as a very good doctor. When he came to Dawson he had been married and was a widower; in Dawson he married a local girl, and

they occupied living quarters adjoining his offices which were over Charles Anderson's store.

The doctor was friendly, well-mannered, and very well liked. He was extremely fond of hunting, and spent much time in the field during the season when it was permissible to hunt prairie chickens. He was a member of the Masonic Lodge.

About 1908 Dr. Triplett removed to Floodwood, where he bought some land, practiced medicine, and engaged in other activities. He was a member of and president of the Board of Education of Floodwood, and at one time was a member of the State Legislature. He had no children. He died December 8, 1922, of myocarditis.

**James Alfred Watson** was born September 5, 1867, in Longford, Ireland. He went to Canada with his father when he was seventeen years of age, and later his mother and three brothers followed. The family settled on a farm at Pilot Mound, Manitoba, and at first James Alfred Watson taught school and thus financed his medical education. He took his medical training at the University of Manitoba, in Winnipeg, graduating in 1894. He began his practice of medicine in Wood Lake, Minnesota, and stayed there four years. He left in 1898 to take up postgraduate work in eye, ear, nose and throat diseases in New York and Vienna between the years from 1899 to 1900. Returning to Minnesota in 1900 he married Lucia Coghlan of Wood Lake and began to practice as an ophthalmologist and otolaryngologist in Minneapolis. He practiced there until his death on June 17, 1939.

During his early years of practice in Minneapolis he was a member of the faculty of Hamline University Medical School.

Beginning with 1920, he practiced as a member of the Eye, Ear, Nose and Throat Clinic, and was associated with Doctors Eugene Strout, John Macnie, and W. E. Patterson. This clinic was located in their own building at 74 South Eleventh Street until the year 1934, and then in the Medical Arts Building.

Dr. Watson became a citizen of the United States soon after starting practice in Minneapolis. He was an active member of the Hennepin Avenue Methodist Episcopal Church. He was also a member of the Hennepin County Medical Society, Minnesota State Medical Association, American College of Surgeons, American and Minnesota Academy of Ophthalmology and Otolaryngology; and a member of the Minneapolis Athletic Club and the Lafayette Club.

Dr. Watson was survived by his widow and four children: Cecil James, Lelan Alfred, Eleanor Isabel, James Alfred, Jr., all of Minneapolis. He was known as a "sound and conservative practitioner of medicine and a true and kindly friend."

Dr. Watson was seventy-two years of age at the time of his death, and lived at 2100 Irving Avenue, Minneapolis. He was past president of the Minnesota Academy of Ophthalmology and Otolaryngology, a member of the Phi Rho Sigma Medical Fraternity and the Automobile Club.

Cause of death was coronary thrombosis; contributory causes, coronary sclerosis, hypertension (three years), apoplexy (one year). Burial was in Lakewood Cemetery.

**Frederick G. Watson** was born in Longford, Ireland. His father and his brother, James A. Watson, came to Canada in 1884, and shortly thereafter Frederick G. Watson, his mother, and two brothers followed. The family settled on a farm at Pilot Mound, Manitoba, and Frederick G. Watson received his elementary

education in the schools of that province. For his medical training he entered Hamline University, graduating about 1895. He also graduated as a pharmacist, and about 1896 went to Wood Lake where he opened a drug business. On September 1, 1897, Dr. Watson married Isabella A. Tibbils of Wood Lake, Minnesota, and shortly thereafter moved to the neighboring town of Clarkfield and began his medical practice there.

Children born to Dr. and Mrs. Watson in the years that followed were: Horace Percy, born October 2, 1898; Leopold Frederick, born November 30, 1902; Sidney J., born August 10, 1904; Donald Goodwin, born October 13, 1907, and Anna Bernice, born August 12, 1909.

In appearance Dr. Watson was very English: tall, rather slender, very reserved and inclined to shyness. His speech was the clipped speech of the Englishman, a characteristic which set him off in a strongly Norwegian community. He had a very brilliant mind and was regarded as a very good doctor. He belonged to the Methodist church and his religious beliefs precluded membership in other organizations.

Clarkfield was a small village of about 320 when Dr. Watson began practice there, and had two Lutheran Churches and a Methodist Church. The language most commonly heard upon the streets at that time was Norwegian. About the same time Dr. Charles Watson, a brother, was practicing at Boyd, Minnesota, and James A. Watson at Wood Lake.

Dr. Watson built up a large practice in the community. He drove long distances to visit patients in the surrounding country, getting about with horse and buggy, or cutter, in all kinds of weather and over roads which were no more than tracks on the prairie sod, or at the best narrow highways which became almost impassible in wet weather. In 1906 his practice had grown so large that he felt it necessary to look for a partner, and the man he thought most suitable was Dr. M. M. Hauge. In 1907 Dr. Hauge came to Clarkfield as Dr. Watson's partner, and the two practiced together for some years. As Dr. Hauge spoke Norwegian fluently, was Norwegian, strongly Lutheran, and with gifts in music and other attributes which made him very popular in this predominantly Norwegian community, it was not long until Dr. Watson found his Norwegian patients preferring to consult Dr. Hauge. When Dr. Watson became convinced that there was no future for him in Clarkfield he left and located in Rushmore, Minnesota, where he stayed ten years. He then went to Worthington and established there a very good practice and a clinic.

Dr. Watson became ill with a heart ailment about 1930, and died January 11, 1932, of a heart attack, at Worthington, Minnesota.

Mrs. Watson survived him, and also his children Horace Percy, Leopold Frederick, Donald Goodwin and Anna Bernice. His son Sidney J. Watson became a doctor, graduating from the University of Minnesota Medical Department, and died prior to his father's death. His daughter Anna Bernice died July 21, 1932. Mrs. Watson died June 11, 1945, at Worthington, Minnesota.

Horace Percy Watson now resides at Sioux Falls, S. D., a partial invalid. Donald Goodwin operates a farm near Redfield, S. D.; Leopold Frederick is a dentist.

*(To Be Concluded)*

## MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

**230 Lowry Medical Arts Bldg., Saint Paul, Minnesota**

**E. M. Jones, M.D., Secretary**

### PHYSICIANS LICENSED FEBRUARY 13, 1953

#### January, 1953, Examination

| *Name* | *School* | | *Address* |
|---|---|---|---|
| ALARI, Heino | Eberhard-Karls U. Tubingen, Germany | MD 1947 | Northwestern Hosp., Minneapolis, Minn. |
| ANDERSON, Hugh Vaux | U. of Oregon | MD 1952 | 3425 S.W. 9th, Portland, Ore. |
| BEDDOW, Ralph Marlowe | U. of Oregon | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| CRISP, Jr., Elmer Ernest | Northwestern U. | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| DAVIS, Charles Truman | U. of Tennessee | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| DENKER, Arthur Gilman | U. of Oregon | MD 1952 | 63 E. Ash, Lebanon, Ore. |
| DUNN, William Joseph | U. of Illinois | MD 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| ELSTNER, Howard L. | U. of Pittsburgh | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| ERICKSON, Robert Dell | U. of Minnesota | MD 1953 | Wykoff, Minn |
| FISHER, Don Harvey | Northwestern U. | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| GALLO, Bela | Peter Pazmany U. Budapest, Hungary | MD 1945 | Lake Bronson, Minn |
| GOSSELIN, Gilles | U. of Montreal | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| HARDY, Paul Edward | St. Louis U. | MD 1951 | Ancker Hosp., St. Paul, Minn. |
| HEALY, John Russell | Jefferson Med. Col. | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| JACKS, Quentin Douglas | U. of Mantitoba | MD 1940 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| JARVIS, Bruce William | U. of Minnesota | MD 1953 | 1869 Marshall Ave., St. Paul, Minn. |
| LEPPO, Niilo Erkki Albert | U. of Helsinki Helsinki, Finland | | 503 Med. Arts Bldg., Duluth, Minn. |
| | Candidate of Medicine | 1931 | |
| | Licentiate of Medicine | 1936 | |
| MacMILLAN, Angus Hugh | Queens U. | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| McPHERSON, Alan Given | U. of Manitoba | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| MANSTEIN, George | U. of Pennsylvania | MD 1941 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| MYERS, Richard Lyon | St. Louis U. | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| OLAVS, Olga | U. of Latvia "Physician" Riga, Latvia | 1942 | 1009 Nicollet Ave., Minneapolis, Minn. |
| PRICE, Mary Lomas | U. of Aberdeen Aberdeen, Scotland | MB SB 1947 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| SATERSMOEN, Jr., Theodore | U. of Minnesota | MD 1948 | Veterans Adm. Hosp., Dearborn, Mich. |
| SIBLEY, John Adams | U. of Chicago | MD 1946 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| SKILLERN, Scott Dorsey | U. of Pennsylvania | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| STREITZ, John McDonald | U. of Minnesota | MD 1952 | Veterans Adm. Hosp., Minneapolis, Minn. |
| VAN PROOYEN, Cornelia Mary | Washington U., Mo. | MD 1951 | Mpls. Gen. Hosp., Minneapolis, Minn. |

#### Reciprocity Candidates

| *Name* | *School* | | *Address* |
|---|---|---|---|
| BEST, Etta Wright | U. of Louisville | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| CARROLL, John Joseph | Creighton U. | MD 1947 | 117½ S. 1st St., Montevideo, Minn. |
| COOK, Clayton Stevens | Tulane U. | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| FELDAKER, Mauri | Washington U., Mo. | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| GOFF, John Robert | U. of Rochester | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| GUNN-SMITH, Allan Murray | U. of Minnesota | MD 1950 | 431 10th Ave., Two Harbors, Minn. |
| HEIST, Erwin William | U. of Iowa | MD 1951 | Jaguum Hosp., Lutheran Mission, Madang, Terr., New Guinea |
| HUGHES, Sidney Osborne | U. of Pennsylvania | MD 1947 | 172 Main St., Winona, Minn. |
| HYMAN, Jr., Orren Williams | U. of Tennessee | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| JESSE, Richard Henry | U. of Nebraska | MD 1949 | Truman, Minn. |
| KEYS, John R. | U. of Cincinnati | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| KUNTZ, Aloysius Leo | Tulane U. | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| LEMMON, Mark Leonard | U. of Texas | MD 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| O'LEARY, James Michael | Jefferson Med. Col. | MD 1946 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| OPFELL, Richard William | U. of Iowa | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| PERRY, Jr., John Francis | U. of Texas | MD 1947 | U. of Minn. Hosp., Minneapolis, Minn. |
| SANDERS, Edward Joseph | Creighton U. | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| SLEDGE, Claire Blount | U. of Oklahoma | MD 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| SPITTEL, Jr., John Allen | U. of Maryland | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |

| *Name* | *School* | | *Address* |
|---|---|---|---|
| TOON, Robert Wallace | U. of Oregon | MD 1944 | 1251 Med. Arts Bldg., Minneapolis, Minn. |
| TROUP, Richard Henry | Northwestern U. | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |

**National Board Candidates**

| *Name* | *School* | | *Address* |
|---|---|---|---|
| ALLEN, George Seaver | U. of Rochester | MD 1950 | Mineral Spgs. San., Cannon Falls, Minn. |
| BAGLEY, Russell Willis | Tufts Med. College | MD 1948 | 871 S. Wilder St., St. Paul, Minn. |
| BEEMAN, Edward Arthur | Boston U. | MD 1947 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| BENJAMIN, Robert Bedell | U. of Minnesota | MD 1952 | 18th & Taylor, Hood River, Ore. |
| BOUCHELLE, McLemore | Harvard U. | MD 1943 | 6th Ave. & 9th St. N., Virginia, Minn. |
| BUCKLEY, Joseph John | New York Med. Col. | MD 1946 | 500 Union St., Minneapolis, Minn. |
| CHAPIN, Lemuel Edward | Temple U. | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| GORDON, Donald Lloyd | New York Med. Col. | MD 1947 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| LIPINSKI, Stanley Wnuk | Northwestern U. | MD 1951 | Hibbing, Minn. |
| SILVER, Lawrence | New York Univ. | MD 1950 | Hosp. of Rockefeller Inst. for Med. Research, 66th St. & N. Y. Ave., New York 21, N. Y. |
| SVIGALS, Robert Elliott | New York Med. Col. | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| VAN BUSKIRK, Charles | Albany Med. Col. | MD 1947 | U. of Minn. Hosp., Dept. Neurology, Minneapolis, Minn. |
| WEBB, Jr., Wilson Davenport | New York Med. Col. | MD 1947 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| YOUNG, John Van Winkle | Northwestern U. | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |

**PHYSICIANS LICENSED MAY 22, 1953**

**April, 1953, Examination**

| *Name* | *School* | | *Address* |
|---|---|---|---|
| AMBRUS, Laszlo | Eberhard-Karls U. Tubingen, Germany | MD 1948 | 462 S. Robert St., St. Paul, Minn. |
| AMBRUS, Milka | Eberhard-Karls U. Tubingen, Germany | MD 1948 | Midway Hosp., St. Paul, Minn. |
| ANDERSON, Jack G. | Marquette U. | MD 1951 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| BALEISIS, Peter | U. of Dusseldorf | MD 1949 | 700 W. Broadway, Minneapolis, Minn. |
| BARLOW, Loren Call | Northwestern U. | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| BOZANIC, Milos S. | Ludwig Maximilian U. Munich, Germany "Arzt" | 1948 | St. Joseph's Hosp., St. Paul, Minn. |
| CONNAUGHTON, Patrick Alex | St. Louis U., Mo. | MD 1951 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| DATESMAN, Robert Wallace | U. of Pennsylvania | MD 1951 | USAH, 3444 ASU, Camp Stewart, Ga. |
| DE WEESE, Jr., Robert Cornell | U. of Colorado | MD 1952 | St. Cloud Nat. Bk. Bldg., St. Cloud, Minn. |
| FEINBERG, Milton | U. of Illinois | MD 1941 | 2215 Plymouth Ave. N., Mpls., Minn. |
| FOLEY, III, Philip James | Northwestern U. | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| GLENNY, Jr., William Robb | U. of Minnesota | MD 1953 | Box 308, Osseo, Minn. |
| GREENWOLD, Warren Eldon | U of Chicago | MD 1946 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| HEPBURN, Allan Lockwood | U. of Alberta | MD 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| JOHNSON, Marvin Waldemar | U. of Minnesota | MD 1953 | Milaca, Minn. |
| KARLINSKY, William | U. of Manitoba | MD 1945 | 644 Niagara St., Winnipeg, Man., Can. |
| KARNOPP, Katherine | U. of Oregon | MD 1952 | Glen Lake San., Oak Terrace, Minn. |
| KONIG, Theodore John | U. of Minnesota | MD 1952 | Veterans Adm. Hosp., Minneapolis, Minn. |
| LASZEWSKI, Franz von Zelberschwecht | Westphalen Wilhelms U. Munster, Germany | MD 1929 | St. Joseph's Hospital, St. Paul, Minn. |
| LYSYJ, Anatol | Ludwig Maximilian U. Munich, Germany | MD 1950 | 624 W. 54th St. S., Minneapolis, Minn. |
| McCULLOCH, Charles Fraser | Queens Univ. | MD 1945 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| MORISSETTE, Gaston | Laval Univ. | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| PLACE, Virgil Alan | Johns Hopkins U. | MD 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| PROSHEK, Lumir Charles | U. of Iowa | MD 1949 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| RICHARDS, Albert MacDonell | U. of Minnesota | MD 1948 | Heart Hosp., U. of Minn., Mpls., Minn. |
| RYZEN, Nicholas | U. of Tennessee | MD 1951 | Anoka State Hosp., Anoka, Minn. |
| SABANAS, Alvina Olga | U. Vytautas the Great Kaunas, Lithuania | MD 1939 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| SMITH, Dale Compton | U. of Kansas | MD 1945 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| SQUIRES, Franklin John | U. of Manitoba | MD 1945 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| THOMPSON, Richard Eugene | Northwestern U. | MD 1951 | NPBA Hosp., Glendive, Mont. |
| TORKELSON, Leonard Beck | U. of Minnesota | MD 1953 | St. Joseph's Hosp., St. Paul, Minn. |
| TSAI, Shih Hao | National Med. Col. Shanghai, China | MD 1941 | Glen Lake San., Oak Terrace, Minn. |
| VOIGHT, Allan Earl | U. of Oregon | MD 1951 | Mpls. Gen. Hospital, Minneapolis, Minn. |
| VREELAND, Oliver Henry | U. of Oregon | MD 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| WESTPHAL, Paul Harry | U. of Minnesota | MD 1953 | Gettysburg, S. D. |
| WOYDA, William Charles | U. of Minnesota | MD 1953 | Box 609, Elk River, Minn. |

## MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

### Reciprocity Candidates

| *Name* | *School* | | *Address* |
|---|---|---|---|
| BOSSARD, John Wesley | U. of Maryland | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| BOYD, George Knox | U. of Pittsburgh | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| BURGERMAN, Arthur | Indiana U. | MD 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| BUTLER, Jr., Lewell Colbert | Louisiana State U. | MD 1946 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| CAYCE, John Hill | U. of Texas | MD 1947 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| CLINGER, Edwin James | Ohio State U. | MD 1946 | 102-110 2nd Ave. S.W., Rochester. Minn. |
| COOPER, Milton | Cornell U. | MD 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| DURAN, Robert Jackson | U. of Oklahoma | MD 1947 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| FYKE, Jr., Frazier Earl | U. of Pennsylvania | MD 1947 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| HUEBERT, Dan W. | U. of Kansas | MD 1946 | 26 Washington, Hutchinson, Minn. |
| JACKSON, Robert Carl | U. of Wisconsin | MD 1945 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| JOHNSON, Robert Hugo | U. of Nebraska | MD 1952 | 210 3rd St. S.W., Little Falls, Minn. |
| KILMER, Warren Lee | Indiana U. | MD 1952 | State Bk. Bldg., Inter. Falls, Minn. |
| LAW, William McConnell | Med. Col. of Va. | MD 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| LEE, Philip Randolph | Stanford U. | MD 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| LOCHRIDGE, Jr., Edwin Payne | U. of Georgia | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| LOWE, James Cecil | U. of Tennessee | MD 1943 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| MacLEAN, Donald Beacom | Wayne Univ. | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| PRICE, Jr., William Edmund | U. of Oklahoma | MD 1946 | 412 Delaware St. S.E., Minneapolis, Minn. |
| PURO, Henry Evert | Stanford U. | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| QUINONES, Rafael Enrique | U. of Tennessee | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| REDELFS, John Wright | U. of Nebraska | MD 1945 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| RICKMAN, James H. | U. of Louisville | MD 1946 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| RYAN, Robert Frank | Stanford U. | MD 1947 | 102-110 2nd Ave. S.W. Rochester, Minn. |
| SCHWARTZ, Carl Alfred | U. of Pennsylvania | MD 1947 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| SCOTT, Jr., Charles | Emory Univ. | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| SMITH, Jr. Fred Newman | Tulane Univ. | MD 1947 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| TAYLOR, Harry Edwin | Temple Univ. | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |

### National Board Candidates

| *Name* | *School* | | *Address* |
|---|---|---|---|
| EDELMANN, Robert Benedict | Long Island Col. Med. | MD 1947 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| GARAMELLA, Joseph John | Georgetown U. | MD 1945 | Veterans Adm. Hosp., Minneapolis, Minn. |
| HARTRIDGE, Virginia Bishop | Woman's Med. Col. of Pa. | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| O'NEIL, Richard Lawson | Boston Univ. | MD 1947 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| WALDMANN, Edward Bernard | Creighton Univ. | MD 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| WANNAMAKER, Lewis William | Duke Univ. | MD 1946 | Dept. Ped., U. of Minn., Hosp., Minneapolis, Minn. |
| WINN, Jr., William Edward | Harvard Univ. | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |

## PHYSICIANS LICENSED JULY 17, 1953

### June, 1953, Examination

| *Name* | *School* | | *Address* |
|---|---|---|---|
| ANDERSON, Richard William | U. of Minnesota | MD 1953 | St. Joseph's Hosp., St. Paul, Minn. |
| ARNESON, Genevieve A. | U. of Minnesota | MD 1953 | U.S.P.H.S. Hosp., New Orleans, La. |
| BAKKE, Arnold Clifford | U. of Minnesota | MD 1953 | St. Joseph's Hosp., St. Paul, Minn. |
| BASINGER, Harold Preston | U. of Minnesota | MD 1953 | Williamsport Hosp., Williamsport, Pa. |
| BEARMAN, Morton | U. of Minnesota | MD 1953 | Ancker Hosp., St. Paul, Minn. |
| BERG, Arnold Martin | U. of Minnesota | MD 1953 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| BOWLIN, Paul Frederick | U. of Minnesota | MD 1953 | Oakland Naval Hosp., Oakland, Calif. |
| BRETZKE, Carl Otto | U. of Minnesota | MD 1953 | Greenbush, Minn. |
| CAMMOCK, Earl Elarth | U. of Minnesota | MD 1953 | Harborview Hosp., Seattle, Wash. |
| CARR, William Johnson | U. of Minnesota | MD 1953 | 338 LaSalle Bldg., Minneapolis, Minn. |
| COUNTRYMAN, Ethlyn | U. of Minnesota | MD 1953 | San Francisco Hosp., San Francisco, Cal. |
| CROUCH, Boyden L. | U. of Kansas | MD 1946 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| DEMARAIS, Francis Emery | U. of Minnesota | MD 1952 | 2508 Colfax Ave. S., Minneapolis, Minn. |
| DOBSON, Mervin Wilbert | U. of Manitoba | MD 1944 | Medical Center, Mankato, Minn. |
| DOXSEE, George Charles | U. of Iowa | MD 1952 | 30 N.W. 1st St., Chisholm, Minn. |
| DUNN, Robert Clarence | U. of Minnesota | MD 1953 | St. Joseph's Hosp., St. Paul, Minn. |
| FILIPOVICH, Orest Nicholas | U. of Minnesota | MD 1953 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| FRENCH, Bayard Taylor | U. of Iowa | MD 1952 | 2229 3rd Ave. E., Hibbing, Minn. |
| FUSARO, Ramon Michael | U. of Minnesota | MD 1953 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| GODFREY, H. Wilson | U. of Minnesota | MD 1953 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| GOODCHILD, William Russell | U. of Minnesota | MD 1953 | Mt. Sinai Hosp., Minneapolis, Minn. |
| GRANQUIST, Richard David | U. of Minnesota | MD 1953 | St. Luke's Hosp., Duluth, Minn. |
| HABERLAND, Lyle Freeman | U. of Minnesota | MD 1953 | Sacramento Co. Hosp., Sacramento, Cal. |
| HAGEDORN, Jr., Max Boykin | Loyola U. | MD 1949 | Veterans Adm. Hosp., Minneapolis, Minn. |
| HALVERSON, William Gearey | U. of Minnesota | MD 1953 | St. Mary's Hosp., Duluth. Minn. |

| *Name* | *School* | | *Address* |
|---|---|---|---|
| HANSON, Daniel James | U. of Minnesota | MD 1953 | Mercy Hosp., Toledo, Ohio |
| HANSON, Harold Walden | U. of Minnesota | MD 1953 | St. Joseph's Hosp., St. Paul, Minn. |
| HEID, James K. | U. of Minnesota | MD 1953 | Madigan Army Gen. Hosp., Fort Lewis, Wash. |
| HEINZERLING, Carl Robert | U. of Minnesota | MD 1953 | Mt. Sinai Hosp., Minneapolis, Minn. |
| HOPPERSTAD, Jule Jerome | U. of Minnesota | MD 1953 | St. Mary's Hosp., Minneapolis, Minn. |
| HOYT, Charles Sherman | U. of Minnesota | MD 1953 | Valley Forge Army Hosp., Phoenixville, Pa. |
| HUFF, John Stephen | U. of Minnesota | MD 1953 | Memorial Hosp., Phoenix, Ariz. |
| JOHNSON, Gordon Erik | U. of Minnesota | MD 1953 | St. Luke's Hosp., Duluth, Minn. |
| JUDE, James Roderick | U. of Minnesota | MD 1953 | Johns Hopkins Hosp., Baltimore, Md. |
| KADESKY, Harold Baird | U. of Minnesota | MD 1953 | 2300 W. 50th St., Minneapolis, Minn. |
| KALLENBACH, Rudolf Werner | U. of Minnesota | MD 1953 | 703 Armstrong Blvd. S., St. James, Minn. |
| KANE, John Francis | Marquette U. | MD 1943 | 117½ S. 1st St., Montevideo, Minn. |
| KAPLAN, Martin Jack | U. of Minnesota | MD 1953 | Michael Reese Hosp., Chicago, Ill. |
| KARON, Everett Howard | U. of Minnesota | MD 1953 | U. of Minn. Hosp., Minneapolis, Minn. |
| KATKOV, Harold | U. of Minnesota | MD 1953 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| KENT, James Roy | U. of Minnesota | MD 1953 | St. Luke's Hosp., Duluth, Minn. |
| KETOLA, Tauno Everett | U. of Minnesota | MD 1953 | Highland Alameda Co. Hosp., Oakland, Cal. |
| KIRSCHBAUM, Thomas Harry | U. of Minnesota | MD 1953 | U. of Minn. Hosp., Minneapolis, Minn. |
| LARSON, Earl Russell | U. of Minnesota | MD 1953 | King Co. Hosp., Seattle, Wash. |
| LENTON, John David | Creighton U. | MD 1953 | Creighton Memorial-St. Joseph's Hosp., Omaha, Neb. |
| LEUZINGER, Donn Ellis | U. of Minnesota | MD 1953 | U.S.P.H.S. Hosp., Seattle, Wash. |
| LINDALL, Dale Regnar | U. of Minnesota | MD 1953 | Tripler Army Hosp., Honolulu, Hawaii |
| LINNELL, Leonard S. | U. of Minnesota | MD 1953 | St. Luke's Hosp., Duluth, Minn. |
| LITIN, Robert Beryl | U. of Minnesota | MD 1953 | Santa Clara Co. Hosp., San Jose, Cal. |
| LITTLE, Bruce Raymond | U. of Minnesota | MD 1953 | Santa Monica Hosp., Santa Monica, Cal. |
| MAGUIRE, Gerald Eugene | U. of Minnesota | MD 1953 | Sacramento Co. Hosp., Sacramento, Cal. |
| MARK, Aaron Louis | U. of Minnesota | MD 1953 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| MARTIN, Jr., George Burnie | U. of Illinois | MD 1950 | 117½ S. 1st St., Montevideo, Minn. |
| McFARLANE, Donald Ray | U. of Minnesota | MD 1953 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| McMAHAN, Robert Oliver | U. of Minnesota | MD 1953 | U. S. Naval Hosp., Oakland, Cal. |
| MEAGHER, Thomas Arthur | U. of Minnesota | MD 1953 | King Co. Hosp. System, Seattle, Wash. |
| MELBY, James Christian | U. of Minnesota | MD 1953 | U. of Minn. Hosp., Minneapolis, Minn. |
| MELLER, Maurycy | U. of Parma, Italy | MD 1937 | First Nat. Bk. Bldg., Brainerd, Minn. |
| MILLER, Harold Dawes | U. of Minnesota | MD 1953 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| MURN, Thomas Gregory | U. of Minnesota | MD 1952 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| NACHTIGAL, Fred C. | U. of Minnesota | MD 1953 | Veterans Adm. Hosp., Long Beach, Cal. |
| NAHAS, Gabriel | U. of Toulouse, France | MD 1944 | 315 Millard Hall, U. of Minn., Minneapolis, Minn. |
| NASH, Eldore Bentley | U. of Minnesota | MD 1953 | Memorial Hosp., Phoenix, Ariz. |
| NEILSON, William George | U. of Illinois | MD 1946 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| NORQUIST, Joseph Louis | U. of Minnesota | MD 1953 | Walter Reed Army Hosp., Washington, D. C. |
| ODLAND, Donald Mark | Marquette U. | MD 1952 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| OLSON, Gregory Mathias | U. of Minnesota | MD 1953 | Sacramento Co. Hosp., Sacramento, Cal. |
| PARSONS, Roger Alan | U. of Minnesota | MD 1953 | St. Joseph's Hosp., St. Paul, Minn. |
| PERKINS, Douglass Everal | U. of Minnesota | MD 1953 | 614 Broadway, Alexandria, Minn. |
| POPADIUK, Peter | Friedrich Alex. U. Erlangen, Germany | MD 1948 | St. Mary's Hosp., Minneapolis, Minn. |
| PRESTON, Frank Stebbins | U. of Minnesota | MD 1953 | St. Vincent's Hosp., New York, N. Y. |
| PUGSLEY, Robert Daniel | U. of Minnesota | MD 1953 | Swedish Hosp., Seattle, Wash. |
| RAMLOW, Ralph Marlyn | U. of Minnesota | MD 1953 | Ancker Hosp., St. Paul, Minn. |
| RICHMAN, Harold Gene | U. of Minnesota | MD 1953 | Barnes Hosp., St. Louis, Mo. |
| RICHTER, David James | U. of Illinois | MD 1952 | 6th Ave. & 9th St. N., Virginia, Minn. |
| ROCKWELL, Winthrop R. | U. of Minnesota | MD 1953 | Tripler Army Hosp., Honolulu, Hawaii |
| RUDIE, William Donald | U. of Minnesota | MD 1953 | St. Mary's Hosp., Duluth, Minn. |
| SCHAFER, Thomas Leo | U. of Minnesota | MD 1953 | Detroit Receiving Hosp., Detroit, Mich. |
| SCHMIDT, Jr., Erwin Rudolph | U. of Wisconsin | MD 1952 | St. Luke's Hosp., Duluth, Minn. |
| SCHNEIDER, James A. | U. of Minnesota | MD 1953 | Gorgas Hosp., Panama, Canal Zone |
| SCHREINER, Leon H. | Northwestern U. | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| SETZER, Jr., Hobert Joseph | U. of Minnesota | MD 1953 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| SHIELDS, Jack William | Stanford U. | MD 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| SISLER, David Allen | U. of Minnesota | MD 1953 | Grand Rapids, Minn. |
| SONTAG, David W. | U. of Minnesota | MD 1953 | Veterans Adm. Hosp., Long Beach, Cal. |
| SUNDBERG, Ruth Dorothy | U. of Minnesota | MD 1953 | U. of Minn. Hosp., Minneapolis, Minn. |
| TAYLOR, Robert Owen | U. of Minnesota | MD 1953 | San Diego Co. Gen. Hosp., San Diego, Cal. |
| TEYNOR, Joseph William | U. of Minnesota | MD 1953 | U. S. Naval Hosp., Great Lakes, Ill. |
| THAL, Sam Harold | U. of Washington | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| TIFFANY, Francis Buchanan | U. of Minnesota | MD 1953 | St. Luke's Hosp., New York, N. Y. |
| ULRICH, Christian Andrew | Northwestern U. | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| VERMUND, Halvor | U. of Oslo, Norway | MD 1943 | U. of Minn. Hosp., Minneapolis, Minn. |
| VORIS, David Clarence | U. of Illinois | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |

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|---|---|---|---|---|
| WALDRON, John Fossen | U. of Minnesota | MD | 1953 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| WALTER, William Edward | U. of Minnesota | MD | 1953 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| WATERS, Alvin W. | U. of Minnesota | MD | 1953 | Ancker Hosp., St. Paul, Minn. |
| WATSON, William Richard | U. of Minnesota | MD | 1953 | Fitzsimons Army Hosp., Denver, Colo. |
| WEGNER, Marwood Emery | U. of Minnesota | MD | 1953 | U.S.P.H.S. Hosp., Staten Island, N. Y. |
| WENZEL, James Brandrup | U. of Minnesota | MD | 1953 | Memorial Hosp., Phoenix, Ariz. |
| WILLIAMS, Paul A. | U. of Minnesota | MD | 1953 | Tripler Army Hosp., Honolulu, Hawaii |
| WILLIAMS, Richard Albert | U. of Iowa | MD | 1952 | Rothsay, Minn. |
| ZELLMER, Richard Edward | U. of Minnesota | MD | 1953 | U.S.P.H.S. Hosp., Baltimore, Md. |
| ZEMMERS, Roberts | U. of Latvia Riga, Latvia | "Physician" | 1926 | St. Luke's Hospital, Duluth, Minn. |

**Reciprocity Candidates**

| *Name* | *School* | | | *Address* |
|---|---|---|---|---|
| BEITHON, Paul Jule | U. of Nebraska | MD | 1952 | Miller Hosp., St. Paul, Minn. |
| BURLEIGH, Jr., Edward Gilbert | Louisiana U. | MD | 1952 | More Hosp., Eveleth, Minn. |
| CALVERT, Wilson Clark | Northwestern U. | MD | 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| COLLINS, Jr., Robert Aaron | U. of Georgia | MD | 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| DYER, John Allen | U. of Pennsylvania | MD | 1947 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| JOHNSON, Morris Blaine | U. of Nebraska | MD | 1952 | Miller Hosp., St. Paul, Minn. |
| LEITSCHUH, Robert Bernard | Marquette U. | MD | 1946 | Sleepy Eye, Minn. |
| LEWIS, Jr., Royce Clay | Tulane U. | MD | 1946 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| McCALL, J. Preston | Baylor U. | MD | 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| McKAY, Walter Edward | Indiana U. | MD | 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| MOBLEY, Jack Ervin | Vanderbilt U. | MD | 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| RACE, George Alexander | Temple U. | MD | 1943 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| SILAS, Ralph Maurice | U. of Michigan | MD | 1943 | Veterans Adm. Bldg., No. 18, Ft. Snelling, St. Paul, Minn. |
| SIMPSON, Joseph Ralph | Louisiana State U. | MD | 1947 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| SMITH, Jr., Ross Haugh | U. of Pittsburgh | MD | 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| SOMMERNESS, M. Duane | Temple U. | MD | 1945 | Willmar State Hosp., Willmar, Minn. |
| THATCHER, Wilbur Clayton | U. of Iowa | MD | 1933 | Physicians Bldg., Fort Dodge, Ia. |
| WALLACE, III, Alexander | Baylor U. | MD | 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| WILLER, Stanley Hermann | Ohio State U. | MD | 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |

**National Board Candidates**

| *Name* | *School* | | | *Address* |
|---|---|---|---|---|
| ALEXANDER, Jr., John Dewey | U. of Pennsylvania | MD | 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| ALTHAUSEN, Jr., Theodore L. | U. of California | MD | 1947 | 65 S. 10th St., Minneapolis, Minn. |
| BRIDGE, Allyn Gustave | Yale U. | MD | 1948 | 301 Kenwood Pkwy., Minneapolis, Minn. |
| DE WALL, Richard Allison | U. of Minnesota | MD | 1953 | 1833 2nd Ave., Anoka, Minn. |
| FARLEY, Frank Giles | Syracuse U. | MD | 1945 | 2229 3rd Ave. E., Hibbing, Minn. |
| GROCH, Sigmund Noel | Tufts Col. Med. Sch. | MD | 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| LINDNER, Janus Casimir | Yale U. | MD | 1950 | 505 Nicollet Ave., Minneapolis, Minn. |
| MARR, Jr., Norval Mason | Cornell U. | MD | 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| NICHOLSON, Richard Waldo | Harvard U. | MD | 1948 | Heron Lake, Minn. |
| RUTLEDGE, John Boyden | U. of Minnesota | MD | 1952 | 1050 Lake Ave., Detroit Lakes, Minn. |
| SKRDLA, Willard Blake | U. of Nebraska | MD | 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |

## PHYSICIANS LICENSED NOVEMBER 13, 1953

**October, 1953, Examination**

| *Name* | *School* | | | *Address* |
|---|---|---|---|---|
| ALLBAUGH, Robert Dean | Washington U., Mo. | MD | 1952 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| AMICK, Lawrence Douglas | U. of Iowa | MD | 1945 | Sac City, Iowa |
| BONNET, John David | Johns Hopkins U. | MD | 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| BOTHAM, Richard James | U. of Wisconsin | MD | 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| BRUNSTING, Jr., Louis Albert | U. of Michigan | MD | 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| CAMPAGNA, Mario Joseph | U. of Oregon | MD | 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| CARPER, John Mark | Jefferson Med. Col. | MD | 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| CHRISTU, Chris Nicholas | Louisiana State U. | MD | 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| COMESS, Morton Seymour | U. of Illinois | MD | 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| CUMMISKEY, Paul Vernon | U. of Minnesota | MD | 1953 | 104 S. 4th St., Mankato, Minn. |
| DEVANNEY, Louis Raynor | Northwestern U. | MD | 1939 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| DOAN, Robert English | U. of Minnesota | MD | 1953 | Mpls. Gen. Hosp., Minneapolis, Minn. |
| DOUGLASS, Jesse Ellsworth | U. of Minnesota | MD | 1952 | 2325 Stinson Blvd., Minneapolis, Minn. |
| HENDRICKSON, Russell Robert | U. of Minnesota | MD | 1953 | Cook Co. Hosp., Chicago, Ill. |
| HIGGINS, John Alden | U. of Minnesota | MD | 1953 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| HOWARD, David Lloyd George | U. of Toronto | MD | 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| HUNTER, Samuel Wynne | U. of Rochester | MD | 1947 | U. of Minn. Hosp., Minneapolis, Minn. |
| JOHNSON, David Arthur | U. of Minnesota | MD | 1953 | Mt. Sinai Hosp., Minneapolis, Minn. |
| LOUGHEED, Lawrence Edward | U. of Manitoba | MD | 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |

## MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

| *Name* | *School* | | *Address* |
|---|---|---|---|
| MAHLE, James Parker | U. of Minnesota | MD 1953 | St. Joseph's Hosp., St. Paul, Minn. |
| McSWEENY, II, Austin J. | Loyola U. | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| MORROW, Jr., George William | U. of Illinois | MD 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| MOULTON, Keith Buell | U. of Minnesota | MD 1953 | St. Joseph's Hosp., St. Paul, Minn. |
| NORDLUND, Mildred Eva | U. of Chicago—Rush | MD 1929 | Cass Lake, Minn. |
| NORMAN, Jr., Mark Lewis | Marquette U. | MD 1952 | 1764 Grand Ave., St. Paul, Minn. |
| OLWIN, Thomas Kent | U. of Oregon | MD 1952 | Ancker Hosp., St. Paul, Minn. |
| OZOLINS, Marta Birzins | U. of Latvia<br>Riga, Latvia | MD 1927 | 274 Iglehart Ave., St. Paul, Minn. |
| PEAKE, Ronald Alan | Indiana U. | MD 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| PITTELKOW, Robert Bernard | Marquette U. | MD 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| POPOVICH, Dragojla, nee'Ostojich | U. of Belgrade<br>Belgrade, Yugoslavia | MD 1939 | Northwestern Hosp., Minneapolis, Minn. |
| RAILE, Richard Bayles | U. of Utah | MD 1950 | 1354 E. Maynard Dr., St. Paul, Minn. |
| RENO, III, George Louis | St. Louis U. | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| SAFIRESCU, Sorin Radu | U. of Bologna, Italy | MD 1950 | Fairview Hosp., Minneapolis, Minn. |
| SEYBOLD, Herbert McCelvey | U. of Texas | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| STEWART, Allan H. | Geo. Wash. U., D.C. | MD 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| SVARE, Gerhart Trygve | U. of Wash., Seattle | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| TAINTOR, Ronald W. | U. of Louisville | MD 1952 | Ancker Hosp., St. Paul, Minn. |
| TERRY, Howard Lincoln | Northwestern U. | MD 1944 | Anoka State Hosp., Anoka, Minn. |
| THOMPSON, Murray Cornwall | U. of Western Ont. | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| TRAYNOR, Jr., Mack Vincent | Northwestern U. | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| VANDEVER, Harry Wallace | U. of Tennessee | MD 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| VEINBERGS, Arnolds | U. of Latvia<br>Riga, Latvia | "Physician" 1940 | Swedish Hosp., Minneapolis, Minn. |
| ZIMERING, Sabina Szwarc | Ludwig Maximilian U.<br>Munich, Germany | MD 1950 | 2741 Chicago Ave., Minneapolis, Minn. |

### Reciprocity Candidates

| *Name* | *School* | | *Address* |
|---|---|---|---|
| BETTS, Charles Samuel | Vanderbilt U. | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| BLATTSPIELER, Lucien H. | U. of Nebraska | MD 1946 | Worthington, Minn. |
| CANTRELL, Glenn | U. of So. Calif. | MD-1947 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| CHRISTIANSON, Herbert B. | Marquette U. | MD 1939 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| CONGDON, Gordon Hall | U. of Chicago | MD 1940 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| CREAMER, Dick Oliver | Baylor U. | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| DeLAND, II, Frank Howard | U. of Louisville | MD 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| ELLIOTT, Dean Cook | Ohio State U. | MD 1946 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| ELLWOOD, Jr., Paul Murdock | Stanford U. | MD 1953 | U. of Minn. Hosp., Minneapolis, Minn. |
| FAUCETT, Robert Lynn | U. of Kansas | MD 1944 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| FONTANA, Robert Scott | St. Louis U. | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| FOX, LeRoy John | Northwestern U. | MD 1945 | NPBA Hosp., St. Paul, Minn. |
| HELMHOLZ, Jr., Henry F. | Johns Hopkins U. | MD 1937 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| HONATH, Donald Herbert | Wayne U. | MD 1947 | 111 W. Main St., Owatonna, Minn. |
| KIRK, Jr., Thomas Allen | U. of Virginia | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| LEAVENS, Milam Edmund | Baylor U. | MD 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| LIMBECK, Donald Arthur | U. of Nebraska | MD 1950 | State Hosp., Fergus Falls, Minn. |
| LOES, Louis Augustine | U. of Iowa | MD 1947 | 1225 St. Germain St., St. Cloud, Minn. |
| MILLER, John William | U. of Louisville | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| MORGAN, Edward Raymond | Tulane U. | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| MUDD, Robert Hiden | U. of Pennsylvania | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| PIFER, Lawrence Harrison | U. of Indiana | MD 1952 | 1218 N. 3rd St., St. Joseph, Mo. |
| REIFSNYDER, III, Wm. Henry | Jefferson Med. Col. | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| RITTER, Donald Glenn | Indiana U. | MD 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| TERRY, Jr., Howard Richards | State U. of N. Y. | MD 1947 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| TURNER, Jr., John Cooper | Tulane U. | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| WALKER, Jr., Harry Charles | Med. Col. of Va. | MD 1951 | U. of M. Grad. Sch., Minneapolis, Minn. |
| WALKER, Jr., Jack Andrew | Southwestern Med. Col. | MD 1948 | 102-110 2nd Ave. S.W., Rochester, Minn. |

### National Board Candidates

| *Name* | *School* | | *Address* |
|---|---|---|---|
| BIRKHEAD, Newton Charles | U. of Pennsylvania | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| BOTTI, John Dominic | U. of Pennsylvania | MD 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| BUIE, Jr., Louis Arthur | U. of Minnesota | MD 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| DOYLE, Jr., John B. | Geo. Wash. U. | MD 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| ENSRUD, Earl Richard | Northwestern U. | MD 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| FABI, Mario Nestor | Geo. Wash. U. | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| FOX, Irwin J. | New York Med. Col. | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| GLASS, Bernard | Ohio State U. | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |

## MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

| *Name* | *School* | | *Address* |
|---|---|---|---|
| GREENFIELD, Duane Leonard | U. of Maryland | MD 1946 | 833 S. Phillips Ave., Sioux Falls, S. D. |
| HADER, Marvin | New York Med. Col. | MD 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| HELME, William Beach | Loyola U. | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| KOGL, Richard Conrad | U. of Minnesota | MD 1953 | Veterans Adm. Hosp., Minneapolis, Minn. |
| LEIGH, James Arthur | Bowman Gray Sch. of Med. | MD 1952 | 606 N. 3rd St., East Grand Forks, Minn. |
| MAGNESS, John Lower | Geo. Wash. U., D. C. | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| MISRAHY, George Albert | Harvard U. | MD 1948 | State Hospital, Hastings, Minn. |
| O'HOLLERAN, Lloyd Stephen | Creighton U. | MD 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| PEMBERTON, Henry Walter | Geo. Wash. U. | MD 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| PETERSEN, Deane Alfred | U. of Nebraska | MD 1946 | 1409 Willow St., Minneapolis, Minn. |
| RANDALL, Alan Douglas | Tufts Col. Sch. Med. | MD 1950 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| ROTHWELL, Walter Spray | Harvard U. | MD 1947 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| SHRINER, John Franklin | Tulane U. | MD 1950 | 1826 Clearmont St., Mobile, Ala. |
| STEINMETZ, Rodney Dunlap | Northwestern U. | MD 1952 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| TWEEDDALE, Duane Newton | Creighton U. | MD 1951 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| UPDIKE, II, Edwin Hoyt | Columbia U. | MD 1949 | 102-110 2nd Ave. S.W., Rochester, Minn. |
| WEISBART, Myron Herbert | Columbia U. | MD 1950 | Veterans Adm. Hosp., Minneapolis, Minn. |
| WILLIAMS, Winfield Tyler | U. of Minnesota | MD 1952 | Veterans Adm. Hosp., Minneapolis, Minn. |

CORRECTION: Owing to an accident in the printing shop, several errors occurred in the list of licentiates as published in the February number of MINNESOTA MEDICINE. The publishers therefore are reprinting the list in its entirety in this issue to rectify the errors which appeared in the previous publication.

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## MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

**230 Lowry Medical Arts Building**
**Saint Paul, Minnesota**

**J. M. Jones, M.D., Secretary**

### FOUR SENTENCED AT MINNEAPOLIS FOR MANSLAUGHTER FOLLOWING ABORTION

*Re: State of Minnesota vs. Willard B. Peterson, Joseph E. Verhota, Vernabell Tuma and Margaret Bishop Bellanger.*

On March 27, 1954, the Hon. Levi M. Hall, Judge of the District Court of Hennepin County, sentenced each of the above named defendants following their pleas of guilty, which they had previously entered, to a charge of manslaughter in the first degree, the charge arising out of the death of a 23-year-old unmarried Minneapolis woman on February 7, 1954, resulting from an abortion performed upon her on January 30, 1954, by the defendant Bellanger.

Mrs. Bellanger, 29, 1116 Como Ave. S.E., Minneapolis, an employe of a downtown bar in that city for the past four years, who had entered a plea of guilty to the charge before Judge Hall on February 16, 1954, was sentenced to a term of not less than five years nor more than twenty years in the Women's Reformatory at Shakopee, the sentence being stayed for five years, however, and the defendant placed on probation for the same period provided she serves the first two years of her sentence in the Women's Workhouse at Parkers Lake.

Following his plea of guilty to manslaughter in the first degree on February 26, 1954, Willard B. Peterson, 37, 226 N. Van Buren, Hopkins, Minnesota, who operates a telephone answering service in Minneapolis, was sentenced by Judge Hall to a term of not less than five years nor more than twenty years at hard labor in the State Prison at Stillwater and his sentence was also stayed for a period of five years, during which time he was placed on probation provided that as one of the conditions of his probation he pays the sum of $1,000 to the Treasurer of Hennepin County within two years.

The defendant Verhota, 38, 7501 Thirteenth Ave. S., Richfield, Minnesota, assistant manager of the same Minneapolis bar referred to above, who also entered his plea of guilty to the manslaughter charge on February 26, before being sentenced by Judge Hall admitted that he had a previous felony conviction in Hennepin County District Court on December 23, 1937, for using an automobile without the permission of the owner. He was then sentenced by Judge Hall to a term of not less than ten years nor more than twenty years at hard labor in the State Prison at Stillwater with the sentence being stayed for three years and the defendant placed on probation for the same period. Vernabell Tuma, 31, 3361 Xenwood Ave., St. Louis Park, Minnesota, employed in the same bar as a hostess, was sentenced to a term of not less than five years nor more than twenty years in the Women's Reformatory at Shakopee with the sentence being stayed for a period of three years during which time she was placed on probation by Judge Hall. Both Verhota and Tuma acted as intermediaries in arranging the abortion.

Mrs. Bellanger, who admitted she had performed a prior abortion in November, 1953, has no medical training or license to practice any form of healing in Minnesota. She did the abortion on January 30 in a Minneapolis hotel room by means of injecting a solution of soapy water and turpentine with a syringe. Peterson, who paid the $500 price for the abortion, turned the money over to Verhota and he kept $150 paying the balance to Tuma. She also retained $150 and paid $200 to Mrs. Bellanger. The young woman who submitted to the abortion was hospitalized subsequently in two Minneapolis hospitals but up until the time of her death she did not furnish the names of any of the defendants to the authorities, contending that the abortion was self-induced. Nevertheless, after her death an investigation disclosed that the facts and circumstances connected with the abortion were as related above.

# Woman's Auxiliary

## DOCTORS' WIVES URGED TO ATTEND DULUTH MEETING

A general invitation to all doctors' wives to attend the annual convention of the Woman's Auxiliary to the Minnesota State Medical Association, June 7, 8 and 9 in Duluth, has been issued by Mrs. C. H. Christensen, president of the St. Louis County Medical Society Auxiliary, hostess auxiliary for the meeting.

The convention is scheduled to coincide, as usual, with the 101st Annual Meeting of the doctors' organization and several important program events, such as the annual banquet and Open House, will be shared by both groups.

The women's program, arranged under the capable direction of Mrs. H. E. Bakkila, with the assistance of Mrs. I. T. Clark, both of Duluth, will begin with registration on Monday morning, June 7, at the Hotel Duluth. The pre-convention board meeting and luncheon will follow at the Kitchi Gammi Club. A tea, arranged through the courtesy of the Art Department of the University of Minnesota's Duluth Branch, will be a feature of Monday afternoon. It will be held from 3 to 5 p.m. at the Tweed Art Gallery, 2531 E. Seventh Street and it will be open to all visiting women. In the evening the women are invited to join their husbands at an Open House and entertainment to be staged in the Ballroom at the Hotel Duluth.

Tuesday's program begins with the annual business meeting and luncheon of the state Auxiliary which is scheduled for the Hotel Duluth. In the evening wives will be guests of their husbands at the annual banquet of the medical organization, also at the hotel. A "round-up" breakfast at the Duluth Women's Club Wednesday morning will bring the meeting to a close.

"Those who are already members of the Auxiliary know the state organization needs their support," Mrs. Christensen said in an appeal to all doctors' wives to attend the Duluth meeting and join in the Auxiliary's work.

"Like any organization, the Auxiliary is made up of individuals—which means you and me.

"We especially wish to urge all doctors' wives who are not already members to take this opportunity to acquaint themselves with the program of the Auxiliary. We feel quite sure they will find a spot in this program in which they would like to serve."

## AUXILIARY CHAIRMAN REPORTS ON MEDICAL EDUCATION FUND

Some interesting and challenging figures, covering medical education in general and its relationship to the American scene, were provided in a report submitted recently by Mrs. J. A. Cosgriff of Olivia, Auxiliary chairman for the American Medical Education Foundation, for Minnesota.

There are seventy-nine medical schools in the United States whose function it is to train doctors to take care of a population of 158 million people, Mrs. Cosgriff points out in this report. These schools produce 0,500 doctors each year at a cost of $10,000 to $12,000 for each doctor. Tuition fees pay for about one-fifth of this amount. Taxes, donations, endowments must cover the rest. Only they failed by an estimated deficit of 19 million dollars, last year, to provide the amount required.

The trouble, according to the report, is that medical training is expensive. It takes 40 per cent of the total University budgets for only 10 per cent of the total number of students. And it is constantly growing more expensive as medical science advances. At the same time, endowments are dwindling, income from endowments has steadily decreased in purchasing power while costs have steadily risen. To complicate matters still further, the population has increased by more than 20 million since 1940 and is currently growing at the rate of 2,500,000 each year.

### Alternatives Are Dangerous

The result for the seventy-nine accredited medical schools is obvious and inescapable. Unless new funds are made available most of them are faced with three equally dangerous alternatives. Either they must lower teaching standards or reduce the number of doctors graduated or accept subsidies from the federal government.

Government subsidy is patently dangerous, says Mrs. Cosgriff's report. To back up the statement she quotes President Eisenhower who said in a statement to Congress that the financial crisis facing medical schools "is a dangerous threat to the national welfare which must be met. The financial problems of medical schools should be solved through private rather than governmental means. Excessive reliance on the government violates the essential principle of our free-enterprise system." Clearly, such reliance could only invite federal domination of medical education.

Compromises in teaching standards, thus brought about, would soon be felt in every community. Local hospital services would deteriorate, family medical care would decline in quality and advances in medical research would be slowed and threatened.

Solution to the whole problem of medical school financing clearly rests upon a privately raised fund on a national level. The American Medical Education Foundation was established for this purpose in 1949, upon recommendation of the medical schools, the American Medical Association and representatives of industry, agriculture and labor. Three objectives were established. They were:

1. To make known the needs of medical education to the American people.

2. To raise annually from private sources the funds necessary to support the medical schools.

3. To distribute these funds equitably to the medical schools.

*(Continued on Page 314)*

# ✦ Reports and Announcements ✦

## THE AMERICAN SOCIETY OF MEDICAL TECHNOLOGISTS

The twenty-second annual National Convention of the American Society of Medical Technologists will be held at Miami Beach, Florida, June 13-17. The co-headquarters hotels are the Deland and DiLido.

## CONFERENCE ON HEALTH IN COLLEGES

Forty national organizations interested in aspects of health and education are joining with the American College Health association in sponsoring the Fourth National Conference on Health in Colleges, May 5-8 in New York. They will consider ways of protecting and improving the health of college students. The theme is "Teamwork in Meeting the Health Needs of College Students."

The conference program will include sixteen working committees in which college and university presidents, deans, physicians, nurses, psychologists, specialists in physical educational and health education, student counselors, and students will pool their knowledge and experience to develop comprehensive health programs integrated with all other college functions.

Previous conferences were held in 1931, 1936 and 1947.

## INDUSTRIAL HEALTH CONFERENCE

The 1954 Industrial Health Conference will be held at the Hotel Sherman, Chicago, Illinois, from April 24-30. The registration fee is $3.00. Reservations are to be made with the Industrial Health Conference Housing Bureau, Hotel Sherman.

April 26-30—Industrial Medical Association

April 25-27—American Conference of Government Industrial Hygienists

April 26-29—American Industrial Hygiene Association

April 27-29—American Association of Industrial Dentists

April 26-30—American Association of Industrial Nurses, Inc.

April 24-30—U. S. Navy I. H. Organization

## EASTERN STATES HEALTH CONFERENCE

The 1954 Eastern States Health Education Conference of the New York Academy of Medicine will be held at the New York Academy of Medicine on Thursday and Friday, April 29 and 30. The program will include four sessions and an evening dinner meeting. The subject of this year's conference will be "Communication in Health Education." A workshop session will be held as part of the education conference and the concepts of communication will be applied to and tested in current health education materials and procedures.

Participation in the conference is limited. Those interested in the conference should mail their requests to Iago Galdston, M.D., The New York Academy of Medicine, 2 East 103 Street, New York 29, New York.

## MEDICAL LIBRARY ASSOCIATION

The Medical Library Association will hold its fifty-third annual meeting June 15-18 in Washington, D. C. The headquarters will be the Hotel Statler, and the official host the Armed Forces Medical Library. The program will include a discussion on medical research by embassy attaches, tours of the National Institutes of Health, the National Naval Medical Center, and of the Armed Forces Medical Library. Delegates to the meeting will hear addresses by Dr. Detlev Bronk, President of the Rockefeller Institute of Medical Research, Lt. Col. Frank B. Rogers, Director of the Armed Forces Medical Library, Mr. Verner Clapp, the Acting Librarian of Congress, and Dr. Raymond Zwemer, Chief of the Library of Congress' Science Division. Further information can be obtained from Lt. Col. Frank B. Rogers, Armed Forces Medical Library, 7th Street and Independence Avenue S.W., Washington 25, D. C.

## MINNESOTA SOCIETY OF NEUROLOGY AND PSYCHIATRY

A regular meeting of the Minnesota Society of Neurology and Psychiatry was held at the Town and Country Club in Saint Paul on March 9. The program consisted of "Clinical experience in the use of succinylcholine chloride (anectine) in electroshock therapy" by Dr. Burtrum Schield and Mr. Philip Margolis.

## ALCOHOL PROBLEMS CONFERENCE AT UNIVERSITY APRIL 1-3

Drinking among young people and drinking and driving are two major topics which were considered at a University of Minnesota conference on the problems of alcohol April 1-3 at the University's Center for Continuation Study.

On the conference program were such outstanding authorities on the alcohol problem as Selden D. Bacon, director of the center of alcohol studies at Yale university; Dr. Paul Stevenson, psychiatrist of the National Institute of Mental Health, United States Public Health Service, Bethesda, Maryland, and the Rev. Forrest L. Richeson of Portland Avenue Christian church, Minneapolis.

The conference was planned by the University and the Minnesota Department of Health for physicians, nurses, welfare workers, judges, teachers, clergymen and other community leaders.

"Prime purpose of this conference," according to William F. Ferguson, chief of the section of mental health and consultant on alcoholism for the state health department, "was to mobilize communities to combat alcoholism. We hope the conference brought to light information about problems of alcohol in our society."

Bacon, a sociology professor who is considered the foremost authority on alcoholism, spoke on such subjects as "Drinking: Customs, Confusion and Conflict," "Nature

and Extent of the Problem of Alcoholism," "Alcoholism—Therapy and Prevention" and "Mobilizing Community Resources to Combat Alcoholism." Bacon and an associate are the authors of the recently published book, "Drinking in College."

Stevenson's subjects were "Personality of the Problem Drinker" and "Alcoholism and the Family."

Rev. Richeson, who has personally counselled about 800 alcoholics in Minnesota, talked about a clergyman's contribution to an understanding of alcoholism.

## HEART DISEASE COURSE AT UNIVERSITY

A course in cardiovascular diseases for general physicians was presented by the University of Minnesota and the Minnesota Heart Association March 22-24. In conjunction with the course, Dr. Samuel A. Levine, clinical professor of medicine, Harvard university, presented the annual George E. Fahr lecture. His topic was "Some Pitfalls in the Care of Cardiacs."

## SCHERING AWARD SUBJECTS ANNOUNCED

To encourage scientific reporting and to stimulate medical students' interest in the ever expanding fields of therapy, the Schering Award Committee has announced the beginning of the ninth consecutive competition in 1954.

Titles of three subjects on which students in the United States and Canada are being invited to submit papers have been made known by George Babcock, Jr., M.D., chairman of the committee. They are:

(1) The Use of Androgen Therapy in the Female

(2) The Prophylactic and Therapeutic Uses of Parenteral Antihistamines

(3) Modern Treatment of Infections and Allergic Disorders of the Eye

For the best paper on each of these subjects, the committee will present one $500 first prize and a $250 second prize. Special citations and professionally useful gifts will also be awarded to all students who submit papers of merit. Outstanding medical authorities in each field will judge the papers.

Deadline for entry forms specifying the student's chosen title is July 1. All manuscripts must be mailed not later than October 1. Students may compete individually or co-operatively in research teams.

The purpose of the Schering Award is to encourage original reporting in the hope that students will later contribute to the essential communication of knowledge throughout the medical profession.

The Schering Award Committee will bring outstanding papers to the attention of editors of appropriate professional journals. Information and instructions for the competition are available from Schering Corporation, 2 Broad Street, Bloomfield, N. J.

## ANATOMY BUILDING NOW JACKSON HALL

A University of Minnesota campus structure, known as the Anatomy building since it was erected forty-two years ago, has been redesignated Jackson Hall by order of the University's Regents. The hall was named for the late Dr. Clarence M. Jackson who became head of the division of anatomy in 1913 and lifted that department to a position of preeminence in the field of medical science.

Dr. Harold S. Diehl of the school of medical sciences announced the naming of Jackson Hall, April 1, at a dinner in the University's Campus club preceding the annual Clarence Jackson lecture. Dr. J. Garrott Allen, professor of surgery at the University of Chicago medical school, delivered the lecture, "Management of Upper Gastro-Intestinal Bleeding."

At the dinner, Elexious T. Bell, University professor emeritus of pathology, reviewed Dr. Jackson's service to the University.

Dr. Jackson, who died at the age of seventy-one on January 17, 1947, gained recognition as one of the first medical school leaders to launch a modern research department. He was acting dean of the graduate school in 1917-18 and again in 1925. He retired in 1941.

Dr. Jackson was editor of Morris Human Anatomy, a widely used textbook, and head of the medical division of National Research Council in Washington, D.C., in 1923-24.

## WOMAN'S AUXILIARY

*(Continued from Page 312)*

The AMEF got off to a slow start but now shows forty-one out of the forty-eight states making substantial contributions to the fund.

The Woman's Auxiliary to the American Medical Association contributed $100,000 from its treasury, and individual state and county auxiliaries gave more than $22,000 in 1953.

### 1954 Contributions Requested

"We auxiliary members have pledged ourselves to help in this most worthy cause," Mrs. Cosgriff reminds the women in this report. "Let us redouble our efforts this year.

"We are still hoping," she says, "that every county auxiliary in the State of Minnesota will make a voluntary contribution to the fund before the year 1954 ends. Earmark your contribution. That is, make a notation on your check indicating whether you wish it to be used for the University of Minnesota, specifically, or if you wish it merely to be added to the general fund. Send it to the treasurer of the State Auxiliary, Mrs. Charles W. Froats, 1406 Edgcumbe Road, Saint Paul, Minnesota."

In that connection, Mrs. Cosgriff notes that "In Memoriam" cards are still available to members, and may be obtained from the state AMEF chairman.

# In Memoriam

## WILFRED P. FRELIGH

Dr. Wilfred P. Freligh, Albert Lea surgeon, died after a brief illness at St. Mary's Hospital in Rochester, February 28, 1954. He was sixty-six years old and had practiced at Albert Lea for the past thirty-four years.

Dr. Freligh was taken ill while he was vacationing in Phoenix, Arizona. He was flown back to Rochester with his wife the day before he died.

He was born in Stillwater and went to school there and at the University of Minnesota, where he received a B.A. degree in 1911 and his medical degree in 1913. He served internships at Abbott Hospital in Minneapolis and at Ancker Hospital in St. Paul. A fellowship followed at the Mayo Clinic in the course of which, he was Dr. Charles H. Mayo's assistant for three and one-half years.

In 1922 Dr. Freligh established himself in Albert Lea and became a member of the staff of the Naeve Hospital of which he was a past-president. He was a lieutenant in the U. S. Army Medical Corps during World War I and was active during his entire career in city, county and state medical groups. He was a member of the Freeborn County Medical Society which he served in a number of official capacities, the Minnesota State Medical Association and the American Medical Association. He was a member, also, of the Alumni Association of the Mayo Clinic, a warden of Christ Episcopal Church of Albert Lea, a past commander of Leo Carey Post of the American Legion, a past director of the Albert Lea Chamber of Commerce, a member of the Elks, of the YMCA and of the Country Club.

Surviving are his wife, the former Irene V. Comstock, and one son, Robert John, 17; also a sister, Mrs. Everett Doran, Sr., of Los Angeles, and a brother, Harold, of Omaha, Nebraska.

## GERALD ARTHUR KNUTSON

Dr. Gerald A. Knutson, well-known Northern Minnesota physician, died suddenly as the result of an embolism, at his home in Hallock, Sunday, March 3, 1954. He was forty-eight years of age.

Death came as the probable consequence of an automobile accident which occured a week earlier as he and his wife were returning to Hallock from Grand Forks where he had gone for a physical examination at a clinic there. Mrs. Knutson suffered several injuries, including probable loss of an eye, and was hospitalized at the time of Dr. Knutson's death. Dr. Knutson, himself, had apparently escaped serious injury and was preparing to go to his office when he was stricken.

He was born at Buxton, North Dakota, the son of Dr. O. A. Knutson, a physician, there, and Mrs. Knutson. He attended the Buxton schools and the University of North Dakota from which he was graduated with B.A. and B.S. degrees. He received his degree in medicine from Northwestern University and served his internship at Ancker Hospital in St. Paul. He practiced medicine in Greenbush for eight years until a fire wiped out his hospital, home and office there. After that he applied for a commission in the Navy and served for three and one-half years during World War II. He held the commission of lieutenant in the Navy when he was discharged and shortly afterwards he went to Hallock to take over the practice of the late Dr. A. W. Shaleen.

Dr. Knutson was widely known in Northern Minnesota. He had recently accepted an appointment to the local board of health of Hallock and he was a member of the Lutheran Church there. He was also a member of the Red River Valley Medical Society, the Minnesota State Medical Association and the American Medical Association.

Surviving are his wife, the former Rush Aaseby, and five children including Carol, now a student at the University of Minnesota, Gerald and Aileen, high school age, Cynthia, a Hallock grade school student, and Richard, below school age. His parents, Dr. and Mrs. Knutson of Buxton survive him, also a brother, Dr. George Knutson, a physician of Negaunee, Michigan, and one sister, Mrs. G. L. Hanson of Mount Vernon, Washington.

## FRED HARWOOD POWERS

Dr. Fred Harwood Powers, Rochester physician for many years, died February 27, 1954, at St. Mary's Hospital in Rochester after a long illness.

Dr. Powers was born in Bloomington, Illinois. He attended high school at Shelbyville, Illinois, and graduated in 1890 from the University of Illinois College of Pharmacy. Later he attended Rush Medical College in Chicago and received his medical degree there in 1900.

He practiced for some years in Illinois, Wisconsin and Ohio and then established himself in general practice, twenty-eight years ago, at 301½ South Broadway in Rochester. He was a member of the Olmsted-Houston-Fillmore-Dodge County Medical Society, the Minnesota State Medical Association and the American Medical Association. He became a life member of the State Association in 1952.

At one time, Dr. Powers operated a small, private hospital in Rochester. He was on the examining board for the air forces, also. He was a 32nd degree Mason, a member of the Order of the Eastern Star, the White Shrine and AOUW.

Surviving are his wife, the former Cora Sprecher, and three daughters, Mrs. Margaret Parish, Chicago; Mrs. George T. Mellinger, New Orleans, Louisiana; and Mrs. Grant H. Elford, Audubon, Iowa. There are five grandchildren.

# Of General Interest

Establishment of an outpatient psychiatric clinic for adults in Saint Paul with **Dr. Clarence J. Rowe** as director of the clinic, has been announced.

The psychiatric center will provide free or part-pay treatment of the mental and emotional ills of persons who are unable to pay the cost of private psychiatric service.

The clinic will accept only patients who are referred to it by physicians, public health agencies, other outpatient clinics, the courts and social agencies.

Recommendation for the establishing of such an outpatient psychiatric clinic for adults came from a committee of doctors, social workers and lay people appointed in June, 1952, by the Saint Paul Area Public Health Council. This committee, headed by Mrs. John Driscoll, made a study of the need for psychiatric services for those persons who are unable to pay the cost of private psychiatric treatment.

The committee report and the establishment of a clinic were approved by the Public Health Council, the practicing psychiatrists in Saint Paul, the executive committee of the Ramsey County Medical Society and the planning and research council of the Saint Paul Community Chest and council.

Location of the clinic has not yet been definitely determined, but it will probably be in a downtown office building. It is hoped that operation can begin about July 1, when Dr. Rowe's appointment takes effect.

Dr. Rowe will head a professional staff that will include a clinical psychologist and three psychiatric social workers. He has been on the staff of the University of Minnesota School of Medicine since 1949, and is an assistant professor in the department of psychiatry and neurology. He has also been attending psychiatrist to the Veterans Administration Hospital for three years and psychiatric consultant for the Family Service of Saint Paul. He is a member of the American Psychiatric Association and the Minnesota Society of Neurology and Psychiatry as well as a diplomate of the American Board of Psychiatry and Neurology.

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**Dr. George N. Kraemer,** formerly of Mankato, has joined the staff of the Thayer-Wandke clinic. Dr. Kraemer is a graduate of the University of Minnesota, a veteran of World War II, and a resident in surgery at Minneapolis General Hospital from 1949-1953.

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**Dr. Allen Hodges,** Albert Lea, and **Dr. Tracy Barber,** Austin, discussed the functions of a mental health clinic and problems in mental health at Neveln PTA in Austin.

**Dr. J. J. Heimark,** Fairmont physician who has practiced in Tri-Mont the past year and a half, is returning to Fairmont to resume his practice in his former office there.

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**Dr. Frances E. Schaar** recently opened her office at sixty-seventh and Lyndale as a pediatrician. She has been with the University hospital since 1947, and is a graduate of the University of Illinois medical school.

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**Doctors C. G. Wingquist** and **A. R. Anderson** have moved into their newly completed office building in Crosby. The one-story structure houses nine rooms on the left of the building and five-room dental office on the right. The exterior has stuccoed sides and a brick front. The lots on either side of the building will provide parking areas for patients.

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**Dr. A. I. Arneson,** Morris, was the speaker for the Father-Son Banquet at the Glenwood Lutheran Church on February 15. Dr. Arneson has made a number of expeditions out of the States hunting big game and he showed one of his motion pictures taken while hunting in British Columbia.

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**Dr. Theodore Bratrud,** Thief River Falls, started his new duties on the staff of the department of pathology, March 1. He will complete his work for a Ph.D. degree in pathology.

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**Dr. F. H. Baumgartner,** Albany, was honored at a testimonial dinner for his ten years of faithful service as mayor of Albany. **Dr. J. B. Beuning,** former Albany physician and surgeon, was the principal speaker.

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**Dr. W. F. Maertz,** formerly of Saint Paul, has been appointed to the staff of the Anoka State Hospital.

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**Dr. Harry M. Weber,** Rochester, was among eighteen on a guest faculty that assisted in the annual postgraduate course in radiology at the University of Kansas medical center in Kansas City in February.

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**Dr. J. K. Butler,** Cloquet, was elected to the executive committee of St. Mary's hospital, Duluth, at their annual staff meeting held on February 4. Dr. Butler will be chief of the department of general medicine at St. Mary's.

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**Dr. Andrew V. Grinley,** Grand Rapids, was elected chief of staff at Itasaca Memorial hospital in February.

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**Dr. and Mrs. C. J. Olson,** Belle Plaine, left Febru-

ary 1 for Temple, Texas, where he took a two-week postgraduate course in heart disease at the Scott Sherwood-Brindley Foundation of the University of Texas Medical School.

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**Lt. Donald S. Amatuzio,** formerly of Duluth, now serving with the U. S. Navy in Japan, recently participated in a unique joint meeting of United States Medical Officers and the Yokosuka and Miura Medical society. Lt. Amatuzio spoke at the meeting attended by 50 Japanese and 100 American doctors. As part of a technical session, Lt. Amatuzio discussed the rapid intravenous dextrose tolerance test. He is a specialist in internal medicine.

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**Dr. C. W. Moberg,** Detroit Lakes, was guest speaker at the February meeting of the Business and Professional Women's Club. His topic was rheumatic fever.

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**Dr. Ralph E. Smith,** Department of Medicine at the Veterans and University Hospitals, Minneapolis, was the guest consultant discussing valvular heart disease at the monthly staff meeting of the Litchfield Hospital.

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**Dr. Beryl Kennedy** of the University of Minnesota Hospital staff meet with the Meeker County Hospital Medical staff at their monthly meeting on March 2. Dr. Kennedy discussed breast cancer with special emphasis on the effect of the use of hormones in its treatment. The clinical conference program throughout Minnesota is sponsored by the Minnesota Department of Health, Minnesota Medical Association, University of Minnesota, Minnesota Heart Association, and the Minnesota Cancer Society.

Volunteer leaders of the American Cancer Society's Minnesota Division, met on February 10 and 11 in Minneapolis. They planned an expanded program of cancer control, education and service. **Dr. Bernard Zimmerman,** Cancer Co-ordinator, University of Minnesota, headed a round-table discussion "The Cancer Problem." Participating in this panel were: **Dr. Marthella J. Frantz, Dr. F. John Lewis, Dr. Donn Mosser,** and **Dr. B. J. Kennedy,** all of the University School of Medicine.

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**Dr. Marguerite Schwyzer,** Saint Paul, suffered a fractured leg in an accident north of Pine City. En route to Miller Hospital, in an ambulance, February 22, they were involved in another collision on Ninth and Main streets in Saint Paul.

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**Dr. Robert L. Pedersen,** who has been associated with **Dr. W. E. Macklin** in the X-ray and radiology department at the Rice Memorial Hospital, Willmar, left for Brainerd, where he will head a similar department at the hospital there.

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**Dr. Frank H. Krusen,** chief of the section on physical medicine, Mayo Clinic, received the President's award for 1953 at Louisville, Kentucky, February 24, during the American Medical association's fourteenth annual Congress on Industrial Health. The award is conferred annually for outstanding contributions to the employment welfare of the handicapped. Dr. Krusen is chairman of the AMA council on physical medicine and rehabilitation.

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**Dr. Martin O. Wallace,** Duluth, spoke at the Duluth Health service annual luncheon meeting on February 25. His subject was "The present status of health insurance plans."

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**Dr. M. Smorazczok,** Monticello, spoke to the PTA in Annandale on February 15. Dr. Smorazczok is a native of Poland; he spent two years in a concentration camp. He spoke on various phases of government under the Communist rule, schools, religion, freedom and other points.

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**William R. Merifield** will be associated with **Dr. Marvin J. Geib,** Moorhead, in his practice of psychiatry as Clinical Psychologist. Mr. Merifield is a graduate of the College of William and Mary with a major in Clinical Psychology. His internship was completed in the Neuro-psychiatric service at Fort Lawton, Washington.

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**Dr. E. J. Engberg,** of the Minnesota School and Colony, Faribault, has satisfied the standards set up by the Advisory Board for Medical Specialties for inclusion in the current edition of the Directory of Medical Specialists. Dr. Engberg has qualified as a specialist in the field of psychiatry and neurology.

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**Dr. and Mrs. H. L. Stemsrud,** Alexandria, left February 26 for a ten-day trip to visit relatives in Milwaukee and to attend a medical conference at the Palmer House in Chicago.

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**Dr. J. L. Delmore, Sr.,** Roseau, was honored with a membership card in the Minnesota Medical Association in the form of an inscribed gold plate in recognition of his 40 years of service in the medical profession.

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**Dr. Paul G. Bauer** has opened an office for the general practice of medicine in Faribault. A graduate of the University of Minnesota School of Medicine, Dr. Bauer took his internship at St. Barnabas Hospital. He served in the Army for three years. His tour of duty included 18 months in Korea as a regimental surgeon.

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Duluth physicians have accepted a goal of $200,000.00 for themselves in the $1,250,000 St. Mary's Hospital Building Fund campaign. **Dr. R. P. Buckley,** chairman of the medical division, made the announcement at the sponsor's committee dinner, February 16. He said Duluth physicians had determined the size of their objective themselves, and had approved it. Teams of doctors will solicit fellow-

practitioners in an effort to reach or surpass the goal.

The Renville County Hospital at Olivia commemorated its third anniversary on February 5. In the period since its opening, the hospital has served 5,356 men, women, and children as in-patients admitted, as well as 1,238 people treated as out-patients. During the same period, 1,725 surgical operations have been done in the hospital and 931 babies have been born. **Dr. J. A. Cosgriff,** Olivia, expressed thanks to the members of the Hospital auxiliary for the service they have rendered to the hospital.

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**Dr. H. P. Van Cleve,** Austin, addressed the Whittier PTA on February 16. He discussed the need for a mental health clinic and the purposes of a family counseling service.

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**Dr. Martin O. Wallace,** Duluth, was the principal speaker at the Duluth Health service's annual dinner. He warned against health plans of too extensive a nature. Dr. Wallace asserted that in England where compulsory plans are extensive abuses have led to lower quality of medical service. "Voluntary, rather than compulsory insurance is cheaper in the long run and maintains a better doctor-patient relationship," Dr. Wallace said.

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Open house was held February 20 in the new clinic building which was opened by **Dr. W. J. Alcorn** in Wabasso. The building includes a waiting room, a room for administration of various types of treatment, the doctor's private and main office, two examining rooms, a pediatric room for care of infants, x-ray room, laboratory room and darkroom. The structure is 31 feet wide and 45 feet long. Construction was begun last summer.

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**Dr. W. D. Cleaves,** Sauk Centre, has moved into new offices in his recently constructed building on Third street.

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**Dr. Tracy Barber,** Austin, was elected president of the Austin YMCA at the kickoff banquet for the 1954 membership enrollment campaign February 17. Dr. Barber is a charter member of the YMCA and a past president of the board of directors. He has been medical director of the George A. Hormel Company since 1948.

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Problems of investigating violent deaths were discussed at the University of Minnesota's coroners' conference March 8. **Dr. Walter Jetter,** professor of legal medicine of Boston university school of medicine, spoke on poisonings, deaths in conflagrations and drownings and suspected drownings. Milton Helpern, assistant professor of forensic medicine, New York university, discussed stabbings and rape, and Briggs J. White of the Federal Bureau of Investigations talked about "trace evidence" and facilities of FBI laboratories.

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**Dr. Richard A. Groat,** a cosultant in the section on anatomy and associate professor of anatomy in the Mayo Foundation, has resigned from the staff of the Mayo Clinic to begin an internship at City Memorial hospital, Winston-Salem, North Carolina.

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Four Mayo Clinic surgeons participated in the scientific program of a sectional meeting of the American College of Surgeons at Omaha, Nebraska, on March 1-4. **Dr. Thomas H. Seldon** took part in a symposium on trauma; **Dr. Frederick A. Figi** participated in a discussion of cancers of the head and neck; **Dr. O. T. Clagett** took part in a symposium on esophagogastric junction; and **Dr. Howard K. Gray** discussed lesions of the pancreas.

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**Dr. Robert B. Pierce** is dividing his time in the practice of medicine between Renville and Olivia.

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**Dr. Byrl Kennedy,** Assistant Professor of Medicine, University of Minnesota, spoke at the first of four clinical conferences for the McLeod County Medical Society in Glencoe on February 25. He discussed new drugs in cancer treatment.

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**Dr. Joseph S. Collins,** Wabasha, is completing his fiftieth year as a physician and surgeon and was made a life member of the Minnesota State Medical Association and the Wabasha County Medical Society.

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**Drs. R. P. Griffin** and **N. W. Wagner,** Benson, announced the formation of a partnership which went into effect March 1. Dr. Griffin, a navy veteran, has been in practice in Benson for seven years. Dr. Wagner became associated with Dr. Griffin about five months ago; previously he had been in Graceville.

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**Dr. and Mrs. C. F. Cervenka,** North Branch, recently attended the Chicago Medical Society's annual clinical conference which was held at the Palmer House from March 2 to 5.

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**Dr. Robert Gray,** Marshall, recently spoke to the Marshall chapter of the University Women at their monthly meeting. He spoke of his experiences in Africa where he spent several years as a physician with the British and American armies and of his findings when he returned later under a Fullbright Scholarship to study native medicines. He told of the problems, politically and socially, that face Africa and those endeavoring to educate and civilize the many tribes there.

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**Dr. Donald E. Dille,** Litchfield, attended the University of Minnesota coroners' conference March 8-10.

The process of incorporating to bring a medical center to Hawley began early in February. Shares of stock are to be sold for $100. Committees have been appointed to inspect various medical centers.

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**Dr. Paul Winchell** was the guest consultant at the first of two clinical conferences for the medical staff at the Montevideo Hospital on March 8. Dr. Winchell of the Department of Medicine at the Heart Hospital on the University Campus discussed case histories on heart disease, particularly emphasizing the types of cardiac diseases that are suitable for heart surgery.

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**Dr. Gaylord Anderson,** director of the school of public health at the University of Minnesota, spoke to the annual mid-winter meeting of the Women's Auxiliary to the Minnesota State Medical association at the International Institute. He discussed the new polio vaccine and gave general information on the work of the physician and how doctors' wives fit into the picture.

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**Dr. Julian S. Ansell,** medical fellow at University hospitals and Veterans hospital, and **Dr. Frank MacDonald,** staff physician at the Veterans Hospital, were guest speakers at a one-day private duty institute of the Minnesota Nurses association February 18 and 19.

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**Dr. John Tobin,** pediatrician, took part in a panel discussion for parents at St. George's Episcopal Church in St. Louis Park.

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Mr. and Mrs. Ivan H. Roberts, Judson, announce the engagement of their daughter, Gloria, to **Dr. Frank J. Carthey,** New Ulm. Dr. Carthey is a graduate of the School of Medicine and the University of Minnesota and is now associated with the Seifert Clinic of New Ulm.

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**Dr. Ivan G. Baronofsky,** assistant professor of surgery at the University of Minnesota, discussed "Research in the Heart Field" at a luncheon meeting of the Exchange club in Saint Paul on February 2.

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**Dr. Robert G. Tinkham,** Minneapolis, was elected vice chairman of St. Barnabas hospital medical staff in February.

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**Dr. Andrew V. Grinley,** Grand Rapids was elected chief of staff at Itasca Memorial Staff in February. **Dr. John Evensta** of Grand Rapids will be assistant chief, and **Dr. R. T. Kelly** of Nashwauk was named secretary. Members of the executive committee for the medical staff are **Dr. O. C. Braun, Dr. Gordon M. Erskine** and **Dr. M. J. Schirber.**

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**Dr. J. K. Butler,** Cloquet, was elected to the excutive committee of St. Mary's hospital, Duluth, at their annual meeting held on February 4. Dr. Butler will be chief of the department of general medicine at St. Mary's.

A conference on sterility and associated problems of reproduction in the human race was presented for physicians at the University of Minnesota center for continuation study in February. Dr. Alan F. Guttmacher, director of the department of obstetrics and gynecology, New York City, and a clinical professor at Columbia, was the guest speaker.

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**Dr. Edward Allen Boyden,** head of the anatomy department in the University of Minnesota medical school, received a special tribute from his friends and associates in observance of his sixty-eighth birthday March 20.

The tribute was the "Boyden Birthday Volume" of *The Anatomical Record,* scientific monthly journal published for anatomists. The special volume consist of four issues of the publication, January through April, 1954. Dr. Boyden, who has been a professor of anatomy at the University since 1931, served as managing editor of the journal for 20 years, 1928-48.

The January issue consists of a short biography of Dr. Boyden, a list of his many publications and five scientific papers contributed by eleven of his colleagues in the anatomy department. Additional papers by former associates will appear in the February, March and April issues.

Dr. Boyden received most of his training at Harvard university where he earned his doctor of philosophy degree in the medical sciences in 1916. He also studied medical anatomy for a year at the University of Freiburg, Germany. He has served on the staffs of six American universities: Harvard, Stanford, the University of California, the University of Illinois, the University of Alabama and the University of Minnesota. His publications include three books, 111 articles in scientific journals and some fifty abstracts and book reviews.

The Minnesota anatomist's studies of the kidneys, gallbladder and lungs have brought him international fame. According to one of his associates, he has made a greater number of important contributions to knowledge of the structure and functioning of the gallbladder and related tubes than any other investigator.

In 1925, he published the first x-ray picture of the normal contraction of the gallbladder in response to a meal of egg yolk. The "Boyden meal" of egg yolk is still a standard test for biliary disease.

During the last eight years, Dr. Boyden has made an intensive study of the segmental anatomy of the lungs. His observations are considered invaluable to the surgeon who wishes to remove a small, diseased part of one lobe of a lung.

His contributions in the field of x-ray diagnosis of gallbladder and lung diseases are the most important of those of any living anatomist—in the judgment of a colleague in the University medical school.

"Through the years, Dr. Boyden has brought illumination to every subject he has investigated," Dr. Charles H. Danforth, professor emeritus of

anatomy, Stanford university, asserted today. "He has been, and continues to be, a potent influence in shaping the quality and the direction of the science of anatomy in America."

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**Dr. Walter Judd** was the principal speaker at a Ninth District Lincoln Day dinner attended by well over 200 people in Warren. **Dr. Ch. H. Holmstrom,** Warren, served as toastmaster.

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**Dr. Ch. H. Holmstrom,** Chairman of the Warren School Board, spoke at the opening of Warren's new gymnasium.

A substantial increase in patients treated at Warren hospital has been noted. Last year a total of 1,842 patients were admitted as compared to 1,603 in 1952, 1,736 in 1951 and 1,697 in 1950. Total number of patient days increased from 8,507 in 1952 to 9,675 last year. The daily average was 26.5 patients.

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**Dr. Donald Steward,** Northwestern Clinic, Crookston, spoke to the members of the Rosseau County Medical Society at the home of **Dr. and Mrs. Lloyd Klefsted,** Greenbush. Dr. Steward spoke on "Bowel obstruction." Several types of tubes for intubation were shown and demonstrated as well as x-rays of actual cases.

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**Dr. O. M. Geilberg** is the new president of the medical staff of the Worthington Municipal hospital. He was named at the annual election meeting of the group held recently. Other officers named include **Dr. D. J. Halpern,** Brewster, vice president, and **Dr. F. L. Shade,** secretary.

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**Dr. W. F. Skaifé** was elected chairman at the recent annual meeting of the Nursing Advisory Board, Little Falls. The board is aiding the nursing service to compile a list of women in the county who are willing to help care for children or old people when assistance is needed.

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**Dr. John F. Kane,** who has been associated with the Hartfiel Medical Center, is leaving Montevideo to become chief surgeon at the Veterans Administration hospital in Miles City, Montana. **Dr. George Martin** and **Dr. J. J. Carroll** who have also severed their connection with the Hartfiel Center are completing arrangements for practice in other cities.

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Officers were elected at the annual meeting of the Upper Mississippi Medical association in Brainerd. Ten counties were represented by 53 doctors. The new officers include **Dr. C. W. Vandersluis,** Bemidji, president; **Dr. V. E. Quanstrom,** Brainerd, first vice-president; **Dr. Luther Davis,** Wadena, second vice president; **Dr. W. Parker, Sebeka,** third vice president; **Dr. G. I. Badeaux,** Brainerd, secretary. Chosen as delegates to the state society were **Dr. Luther Davis,** Wadena, and **Dr. A. M. Watson,** Royalton. Alternates are **Dr. William Anderson,** Brainerd, and **Dr. Douglas Johnson,** Little Falls.

Failure to report several cases of less serious communicable diseases has resulted in a very healthy appearing annual health report for the city of Fairmont, **Dr. M. H. Marker,** city health officer, said as he turned over his report to the city council at its February meeting. The report shows that the city had three cases of polio, two of typhoid fever, one of tuberculosis and one of scarlet fever.

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Quarrelling in children is not all bad, **Dr. Robert Faucett,** psychiatrist and a member of the Mayo Clinic staff, told members of the parents group of the Aldrich Memorial Nursery school. He said that there are some good and constructive things about sibling rivalry in that it can teach a child how to get along in a competitive world. Dr. Faucett was moderator for a panel discussion on "Sibling Rivalry and Jealousy in Children." Also taking part was **Dr. H. F. Helmholz, Sr.,** emeritus member of the Mayo Clinic staff in the pediatrics section; he pointed out the importance of trying to see a situation from a child's point of view and emphasized that parents must avoid making children jealous by comparing their accomplishments with others.

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**Dr. and Mrs. H. J. Elliott** of the Hayfield Medical Center showed slides of their work as medical missionaries in Africa at a meeting of the Kasson PTA in February.

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**Dr. Grace Roth** of the Mayo Clinic and associate professor in physiology of the Mayo foundation spoke on "You and Your Heart" at a dessert meeting of the Scandinavian club February 22 at the YMCA in Rochester.

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A plan which would make cancer tests available to everyone in Minnesota was explained by **Dr. David P. Anderson,** Austin, president of the Minnesota Cancer Society, at a meeting of the volunteer cancer workers. In his speech Dr. Anderson said that under the program sponsored by the Minnesota State Medical Association, doctor's offices would be made cancer detection centers. Patients would undergo the tests designed to detect cancer before any symptoms appear, as a part of their yearly physical examinations.

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The Medical Staff of the Lutheran Hospital held its business meeting February 17. The following officers were elected: **Dr. Harry Palmer,** Blackduck, president; **Dr. John Griffin,** vice president, and **Dr. D. D. Whittemore,** secretary-treasurer.

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Dr. L. R. Gowan spoke to the St. Mary's Council on Volunteers, Duluth, on the subject "Psychiatry in the general hospital."

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Two University of Minnesota surgeons and a former member of the University medical school pediatrics staff have been awarded grants of $30,000 each

by the John and Mary R. Markle Foundation of New York which has named them "Scholars in Medical Science." Announcement of the awards was made by John M. Russell, executive director of the foundation.

The Minnesota scholarship winners are: **Dr. Gilbert S. Campbell,** 829 25th Avenue S.E., Minneapolis, resident in surgery, and **Dr. Mitchell W. Spellman,** 2021 Commonwealth Avenue, Saint Paul, assistant professor of surgery and research fellow. Both are on the staff of Dr. Owen H. Wangensten, University surgery chief.

Also named a Markle scholar was **Dr. Robert A. Ulstrom** who left the University's pediatrics department staff last September to become an assistant professor of pediatrics at the University of California school of medicine, Los Angeles.

The Markle grants are being awarded this year to twenty-five faculty members of medical schools in the United States and Canada. Each grant is awarded at the rate of $6,000 annually for five years, according to Russell, "to offer both academic security and financial help to faculty members at the start of their careers in academic medicine. They (the twenty-five) were selected from sixty candidates nominated for the grants by deans of medical schools."

Four other University of Minnesota doctors received the scholarships in previous years. They are **Dr. George E. Moore,** 1949, then instructor in surgery and now director of Roswell Park Memorial Cancer institute at Buffalo, N. Y.; **Dr. Robert A. Good,** 1950, associate professor of pediatrics; **Dr. Leonard F. Peltier,** 1952, assistant professor of orthopedic surgery; and **Dr. William F. Scherer,** 1953, assistant professor of bacteriology.

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## BLUE SHIELD-BLUE CROSS NEWS

Of increasing interest is the statistical data compiled annually which shows the utilization of Blue Shield and the number of services furnished Blue Shield subscribers by doctors of medicine. On a comparative basis, each type of service in 1953 increased numerically over the number in 1952 and the dollar payment for each service also showed an increase. Such services as in-hospital medical care, emergency medical care, obstetrical, surgical and ancillary services show a marked increase both in the number of cases in 1953 over 1952, and in the amount paid by Blue Shield for each of such services to doctors of medicine.

During the year 1952, the average payment made to doctors for in-hospital medical care was $22.80 per case. In the year 1953, this amount increased to $24.66 per service while the number of such cases increased 13 per cent over 1952. More remarkable is the increase in emergency medical care payments, and the increase in the number of such cases compared with the year 1952. Payments during the year 1952 for emergency medical care average $31.01 per case. However, in the year, 1953, this payment increased to $38.38 per case or an increase of $7.37 more than the amount paid per case in 1952. In addition, the number of such cases of this nature almost doubled.

Obstetrical services furnished Blue Shield subscribers have always been one of the more frequent of Blue Shield services, and while the cost per case in 1953 was approximately the same as the cost in 1952, the number of such cases increased 8.5 per cent over 1952. The cost per case shows the amount paid by Blue Shield to be greater than the scheduled allowance for a normal delivery, but this is clarified by the fact that included in the figures are the higher benefits for cesarean deliveries. This increased cost of obstetrical cases will further increase in 1954 since the Blue Shield allowance for routine obstetrical deliveries has been raised from $50 to $60.

While surgical services rendered Blue Shield subscribers showed an increase of 14 per cent over the number in 1952, the average service payment in 1953 remained practically the same as that of 1952.

Another classification showing marked increases are those known as ancillary services. These include anesthesia, x-ray and endoscopic services. Each increased over 1952. Anesthesia cases, for example, increased in number 31.6 per cent over 1952 and the cost of anesthesia services was 33 per cent greater than in 1952. X-ray services increased in number and the cost increased correspondingly. A greater increase was noted in the number of endoscopic services which amounted to 26 per cent above that of 1952, and the cost of endoscopic services was slightly greater per case in 1953 than in 1952.

It is apparent from these analyses that in both the number of services and the amounts paid for the different services, Blue Shield service to subscribers and doctors has grown appreciably in 1953 as compared with 1952. With the increase of fees or benefits which became effective for services rendered on and after January 1, 1954, there is a great likelihood that the increases in both volume and costs will be even greater in 1954.

Blue Cross subscribers and their dependents used more hospital care in 1953 than in any year in the history of Minnesota Blue Cross. A total of 167,484 participant subscribers . . . one out of every six . . . were hospitalized and over fifteen million dollars was provided for their care, an increase of approximately 2½ million dollars over 1952.

The record breaking number of hospital cases paid during the year (1953) represented an increase of 14 cases per year per 1,000 contracts protected over the previous year.

This increase, it was reported, occurred in the number of bed-patient cases . . . 144,006 participant subscribers received bed-patient care during 1953 compared with 142,372 participant subscribers in 1952. The average length of hospital stay for bed-patient care during 1953 was 6.5 days compared with 6.2 days in 1952.

Out-patient care during the year was given to 23,478 participant subscribers compared with 24,673 during 1952. This increase in bed-patient care and decrease in out-patient services resulted in an increase in the

*(Continued on Page 322)*

# BOOK REVIEWS

**Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.**

THE ANATOMY AND SURGERY OF HERNIA. By Leo M. Zimmerman and Barry J. Anson. 350 pages. Illus. Price $10.00. Baltimore: Williams and Wilkins, 1953.

This new publication dealing with various types of hernia places major emphasis on the inguinal hernia. However, it deals with all types of hernia, and the discussion of each is thoroughly satisfactory. The anatomical section is thorough and meticulously carried out. The section on surgical technique covers diagnosis, preoperative preparation, choice of anaesthetic and detailed description of surgical technique. The authors' ideas in dealing with hernia differ somewhat from the accepted concepts; however, their ideas are good and are well founded. The book loses a great deal of its effectiveness because of needless repetition. The reader is belabored with details of anatomy which are presented adequately in other parts of the book.

The book is a worthwhile addition to the general surgical library.

T. M. OWENS, JR.

PLANNING GUIDE FOR RADIOLOGIC INSTALLATIONS. By Wendell G. Scott, M.D., chairman of the committee on planning of radiologic installations of the commission on public relations of the American College of Radiology. 336 pages. Illus. Price $8.00. Chicago: The Year Book Publishers, Inc., 1953.

With the marked advances of x-ray in the fields of diagnoses and therapy, the radiologist and his associates have found a place of increased importance in the field of clinical medicine. Slowly, but inevitably, the x-ray department has emerged from its few dusty rooms in the basement to become the very center of the community hospital. Because of the broad use to which x-ray has been put and because of its extensive diagnostic and therapeutic contributions, the department of radiology has become the meeting place for physicians in the hospital; a place for them to congregate and discuss their cases with the radiologist and other colleagues. Planning and innovations in the design of radiologic installations have not kept pace with the rapid growth of this specialty. In certain geographic areas there is evidence of originality of design in the functional arrangement of the departments, but these are few and, only too frequently, the radiologist has had no part in the planning and arrangement of a department of which he is to assume direction.

In this authoritative guide, the viewpoints of radiologists, physicists, architects and hospital administrators have been assembled into one exhaustive manual for the first time. Consideration has been given to every type of installation, from a small office to a large hospital department. Adequate space is devoted to diagrams and ideas for the flow of all activities in the radiology department so as to achieve "the most effective traffic pattern for the radiologist and professional staff so that they may interview, examine and treat patients as quickly and thoroughly as possible." Of marked importance are the data for adequate protection of a radiological installation for the radiation worker as well as anyone working in adjacent areas.

This book is invaluable to those persons responsible for the installation of any type of x-ray equipment, be he a general practitioner, a dentist with a single x-ray unit or the director of a large x-ray department.

J. F.

BIBLIOGRAPHY OF OCCUPATIONAL HEALTH. Public Health Service Publication No. 300. Washington, D. C.: Government Printing Office, 1954.

A comprehensive bibliography of Public Health Service publications on occupational health subjects issued through 1953 has just been released by the U. S. Public Health Service.

The purpose of this bibliography is to provide a complete and continuing record for reference use. It calls to the attention of the readers, particularly those new to occupational health, voluminous information on early pioneering studies and other contributions by the Public Health Service to the detection and control of occupational diseases. At the same time, by reflecting changes in subject matter from year to year, it affords a historical perspective of shifting interests in the field of occupational health and serves as an index of progress.

While the limited supply lasts, sample copies of Public Health Service Publication No. 300, "Bibliography of Occupational Health," can be made available from this Division upon request to persons and organizations concerned with health problems in industry. Copies may also be procured from the Superintendent of Documents, Government Printing Office, Washington 25, D. C., for thirty-five cents each.

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## BLUE SHIELD-BLUE CROSS NEWS

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average lengtth of hospital stay for all Blue Cross cases from 5.4 days in 1952 to 5.7 days in 1953.

Every twenty-four hours during 1953 Blue Cross participant subscribers used 2,612.3 days of hospital care compared with 2,472.4 days of care during 1952, 543,493.2 days of hospital care were provided throughout the year 1953 compared with 904,901.6 days of care in 1952, an increase of approximately 50,000 days of hospital care.

Every nine hours during 1953, a Blue Cross participant subscriber incurred a hospital bill totalling $1,000 or over, as compared with one every thirty-nine hours during 1952. During the last quarter of 1953, hospital bills of $1,000 or over were incurred every eight hours.